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Examination of a Method for Increasing Adjustment to and Compliance With Nasal Continuous Positive Airway Pressure (CPAP) in Persons With Obstructive Sleep Apnea Syndrome.

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**EXAMINATION OF A METHOD FOR INCREASING ADJUSTMENT TO AND
COMPLIANCE WITH NASAL CONTINUOUS POSITIVE AIRWAY PRESSURE
(CPAP) IN PERSONS WITH OBSTRUCTIVE SLEEP APNEA SYNDROME**

**A Dissertation
Submitted to the Graduate Faculty of the
Louisiana State University and
Agricultural and Mechanical College
in partial fulfillment of the
requirements for the degree of
Doctor of Philosophy**

in

The Department of Psychology

**by
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Abstract

Obstructive sleep apnea syndrome (OSAS) is a serious medical condition that occurs during sleep and consists of episodes of complete (respiratory pauses) or partial obstruction (hypoventilation) of the upper airway. Approximately 80% of persons diagnosed with OSAS are prescribed nasal Continuous Positive Airway Pressure (CPAP) treatment, which has proven to be the treatment of choice for OSAS. However, noncompliance with CPAP treatment in OSAS patients is a widely recognized problem, and many persons refuse CPAP as a treatment option or fail to use it reliably. Investigations of CPAP use in OSAS patients have generally found that nightly use averages less than five hours. Few interventions have been scientifically evaluated for improving CPAP compliance. The current study evaluated a method of introducing OSAS patients to CPAP prior to the administering CPAP titration in the laboratory. Participants in the treatment groups underwent a 30-minute CPAP habituation trial, with a range of pressures, prior to the polysomnography with CPAP. It was hypothesized that the participants who experienced CPAP habituation would have better sleep quality during CPAP, would be more likely to accept CPAP, and would use CPAP more on a nightly basis than control participants who experienced the usual laboratory procedures for introducing CPAP (CPAP education) to OSAS patients. There were no statistically significant differences for any of the dependent variables between participants who experienced CPAP habituation and participants who experienced CPAP education. Men were found to use CPAP 1.61 hours more on a nightly basis than women ($p = .03$). This difference is most likely attributable to severity, as men were observed to have an A+HI that was twice the observed A+HI of women participants. Overall, CPAP

acceptance and compliance for the complete sample was comparable to what has been reported in the CPAP treatment literature.

Introduction

Obstructive sleep apnea syndrome (OSAS) is a serious medical condition that occurs during sleep and consists of episodes of complete or partial obstruction of the upper airway, which coincides with excessive daytime sleepiness (EDS; Orr, 1997). It is the most common organic disorder of EDS that is diagnosed in sleep disorders clinics and is the most common of the sleep apnea syndromes (Partinen, 1994). Furthermore, OSAS is the most severe and most common form of sleep apnea/hypopnea syndrome (SAHS) and can have dire health consequences if not properly managed. Central sleep apnea is also a serious sleep-related breathing disorder during which repeated respiratory pauses occur during sleep, presumably due to a loss of respiratory effort (White, 1994). Unfortunately, no completely effective treatment for central sleep apnea has been developed, and the focus of the present review will be on OSAS and its treatment. Cross-sectional studies indicate that the minimum prevalence of SAHS is at least 1% (Partinen, 1994). However, the prevalence of SAHS increases with age, and a recent study of persons aged 30 to 60 years found rates of sleep apnea of 2% for women and 4% for men (Young et al., 1993). Additionally, these same investigators found that 4% of women and 9% of men aged 50 to 60 had more than 15 apneas per hour asleep (Young et al., 1993). In persons aged 65 and older, as many as 25% may experience more than 5 apneas per hour asleep (Kripke & Ancoli-Israel, 1983). Of those diagnosed with OSAS, it is estimated that 80% are prescribed nasal Continuous Positive Airway Pressure (CPAP) treatment. Nasal CPAP has proved to be the treatment of choice for patients with OSAS due to its great effectiveness in eliminating apnea and normalizing the sleep architecture (i.e., the composition of sleep; Sullivan & Grunstein, 1994).

In OSAS, episodes of complete obstruction of the upper airway are referred to as apneas while episodes of partial obstruction are referred to as hypopneas. An episode of complete (i.e., apnea) or partial airway obstruction (i.e., hypopnea) must last for a minimum of 10 seconds and must occur during sleep to be scored as a respiratory event. However, sleep apneas and hypopneas typically last between 20 and 40 seconds, and severe events can last up to 120 seconds. These sleep-related respiratory events are usually accompanied by a drop in available blood oxygen (SaO₂) and often by a central nervous system (CNS) arousal response and an autonomic nervous system (ANS) arousal response. Such activation of the CNS results in transient arousals from sleep, and if great enough, complete awakenings. Transient arousals from sleep take the form of bursts of alpha waves (8-11 cycles per second) lasting between 3 and 14 seconds, which degrade the overall quality of sleep causing it to be light and discontinuous (American Sleep Disorders Association [ASDA], 1992). If transient arousals occur at high rates, the sleep architecture can be markedly altered, with reductions of stages 3 and 4 non-rapid eye movement (NREM) sleep, referred to collectively as slow wave sleep (SWS) and a reduction of rapid eye movement (REM) sleep. Large numbers of transient arousals will also increase stage 1 NREM sleep, which is regarded as light/transitional sleep. Blood oxyhemoglobin desaturation usually occurs during or shortly after sleep apneas and hypopneas, with oxygen desaturation below 85% being considered severe, although desaturations can be as low as 50%.

One important measure of the severity of OSAS is the number of sleep-related respiratory events per hour asleep, which is given in the form of the Apnea + Hypopnea Index (A+HI). The diagnostic criterion for the frequency of sleep-related respiratory events in SAHS is a minimum of 5 events per hour asleep; however, significant

pathology is not thought to occur until there are 10 or more events per hour asleep (Guilleminault, 1994). Patients suffering from clinically significant symptoms and sequelae of OSAS typically will have 20 or more episodes of apnea/hypopnea per hour asleep. People suffering from severe OSAS may have apneas with arousal as often as twice per minute, or 120 arousals from sleep per hour (Roth, Roehrs, & Zorick, 1982). According to the authors of the International Classification of Sleep Disorders-Revised, in addition to the criterion of an A+HI of at least 5 per hour asleep, the respiratory obstructions must be accompanied by one or more of the following: frequent arousals from sleep that are associated with airway obstruction, bradycardia, and arterial oxygen desaturation that is associated with the sleep-related respiratory events (ASDA, 1997). Severity of OSAS is categorized as mild, moderate, or severe according to the A+HI, the level of daytime sleepiness, the severity of oxygen desaturation, the presence of cardiac arrhythmias, and the level of impairment in daytime functioning (ASDA, 1997). In general, the daytime consequences of OSAS are correlated with the severity of the apneas (Roehrs, Zorick, Wittig, Conway, & Roth, 1989). Reports of subjective sleepiness and alertness may vary between patients with similar levels of objective severity; however, a clinical interview will often reveal unusual sleepiness, with patients reporting that they fall asleep during a variety of activities (Guilleminault, 1994). Often, the development of sleepiness in OSAS is insidious with patients presenting for treatment after they have endured symptoms for a number of years (Orr, 1997).

The most common presenting symptom in adults with OSAS is EDS (ASDA, 1997). This symptom may be more evident to persons with OSAS or to observers because the perception of sleepiness varies across individuals with reported sleepiness

not always being congruent with objective measures of sleep propensity. The degree of impairment in daytime functioning is variable and ranges from marked impairment due to irresistible urges to sleep to only moderate daytime sleepiness, which is usually unmasked only during periods of low stimulation. Patients with EDS may fall asleep during low stimulation activities such as watching television or during meetings. In severe cases of EDS, patients will report falling asleep involuntarily while eating, talking, or performing very important activities such as driving. The presence of EDS can be of particular importance in occupations that require sustained attention, high levels of concentration, or repetitive tasks (e.g., long-haul truck driver or nuclear power plant operator). The overwhelming sleepiness experienced by many patients with OSAS has been shown to result in a higher frequency of occupational and driving accidents as compared to the general population (Guilleminault, 1982; Roth, Hartse, Zorick, & Conway, 1980). Sleep fragmentation and sleep deprivation from OSAS can result in episodes of automatic behavior during which the affected person engages in semi-purposeful but inappropriate behavior during brief episodes of involuntary sleep referred to as microsleeps (Aldrich, 1994). Loud and persistent snoring (i.e., vibration of the soft palate and posterior faucial pillars due to increased airway resistance in sleep) is the most common cause of referral for OSAS (Guilleminault, 1994). Snoring in OSAS patients is often interrupted by silences (absence of airflow), which are followed by loud snorting or gasping sounds and large body movements when breathing is resumed (Guilleminault, 1994). If a respiratory event is severe, it is not unusual for the affected person to raise the upper body and partially sit up in bed at the termination of the event, and such loud snoring and gross body movements frequently result in the spouse sleeping in a separate bedroom (Guilleminault, 1994). Other symptoms of sleep

apnea include frequent nocturnal awakenings accompanied by sensations of choking or not being able to breathe, reduced energy, significantly reduced sleep onset latencies, frequent morning headaches, diminished ability to concentrate, mild memory impairment, and increased irritability from sleep fragmentation and partial sleep deprivation (Guilleminault, 1994). Additionally, upper airway obstruction is associated with changes in esophageal and gastric pressures, which produces complaints of heartburn and acid reflux in some patients with OSAS (Guilleminault, 1994). Nocturia is a common symptom also with 28% of patients reporting at between 4 and 7 trips to the bathroom per night (Guilleminault, 1994). Lastly, impotence or lack of sex drive has been noted in approximately 28% of patients with OSAS (Guilleminault, 1994).

Certain medical conditions are associated with OSAS, and certain drugs can influence the severity of OSAS. Obesity (i.e., being 20% or more above ideal body weight) is present in approximately two thirds of patients with OSAS (Guilleminault, 1994). Additionally, cardiovascular disease and arterial hypertension are clearly associated with OSAS (Shepard, 1994). CNS depressant drugs can exacerbate the severity of OSAS, and alcohol ingested close to bedtime has been shown to increase the number of complete obstructive apnea events and to prolong their duration (Issa & Sullivan, 1982; Scrima, Broudy, Nay, & Cohn, 1982). This phenomenon holds true for hypnotics and tranquilizers if taken close to bedtime because they have effects similar to alcohol (Sanders, 1994).

Assessment of OSAS

Approximately 74,600 patients undergo polysomnography in accredited sleep disorders centers each year, and about 75% of these patients are diagnosed with OSAS (American Thoracic Society [ATS], 1994). Although presenting complaints of loud

snoring and EDS may suggest the presence of OSAS, a diagnosis can only be made after the patient's sleep is studied objectively by physiological means, which is usually conducted in a sleep laboratory. A nocturnal diagnostic polysomnography (PSG), or sleep study, is the best clinical method for diagnosing the presence of OSAS.

Polysomnographic assessment of OSAS should include central and occipital measures of electroencephalogram (EEG), electrooculogram, electromyogram (EMG), nasal/oral airflow, respiratory muscle effort, electrocardiogram, and blood oxygen saturation (pulse oxygen; ATS, 1994). The physiological recording of respiration during an episode of sleep apnea reveals that airflow ceases or is greatly reduced periodically, which is evident at the level of the nostrils and mouth, while strain gauges encompassing the patient's chest and abdomen register the continuous respiratory effort of the diaphragm and intercostal muscles. Transient arousals resulting from respiratory events (apneas or hypopneas) are apparent in the EEG and EMG channels as bursts of phasic activity that are easily discernible from baseline. A multiple sleep latency test (MSLT) can be used to determine the extent of excessive daytime sleepiness, though this is not essential for diagnostic assessment. The MSLT is an objective measure of daytime sleepiness and consists of a series of four or more nap opportunities, which are scheduled for every two hours beginning two hours after a night of sleep in the laboratory (Carskadon & Dement, 1977). The measure of daytime sleepiness for the MSLT is the sleep onset latency, with values five minutes or less indicating pathological sleepiness.

Pathophysiology and Mechanics of Upper Airway Resistance

Resistance of airflow during sleep exists on a continuum from that which is detectable only with special diagnostic equipment, to mild snoring, to a range of severe

blockages of the pharynx as occurs during SAHS (Guilleminault, 1994). Factors such as the decreased upper airway patency, that is hypothesized to stem from changes in neuromuscular control brought about by sleep (e.g., hypotonicity), and various anatomical abnormalities of the structures surrounding the airway (e.g., excess fatty tissue, stenosis of the upper airway, maxillofacial abnormalities) result in increased resistance of airflow in the upper airway (Isono & Remmers, 1994). Moreover, resistance of airflow in the upper airway during wake has been found to be greater in patients with OSAS than in control subjects (Anch, Remmers, & Bunce, 1982).

Although the upper airway consists of the extrathoracic trachea, the larynx, the pharynx, and the nose, the pharynx is the only segment of the respiratory tract that is capable of collapsing during sleep apnea (Isono & Remmers, 1994). The pharynx is a specialized portion of the upper airway and has additional functions other than conducting gases to and from the lungs. This structure is involved in swallowing and phonation, and by necessity is a highly flexible structure under sophisticated CNS control (Isono & Remmers, 1994). During wakefulness, pharyngeal patency is maintained by continual neuromuscular control of the CNS and by a balance of internal and external airway pressures. In the absence of full neuromuscular control of the upper airway, such as occurs in sleep (especially REM sleep), the pharyngeal lumen (the total cross-sectional area that gases may pass through) of the passive pharynx is influenced to a greater degree by the balance of internal and external pressures sources. The tube law for the passive pharynx model states that the size of the pharyngeal lumen is determined by opposing pressure forces located within and external to the upper airway (Isono & Remmers, 1994). The absolute effect of these forces on the size of the pharyngeal lumen in the absence of muscular activity is referred to as the transmural

pressure of the passive pharynx and is determined by a balance of luminal pressure (i.e., outward lateral pressure acting on the internal pharyngeal wall) and tissue pressure compressing the external wall of the pharynx, which comes from a variety of sources (e.g., large tongue, gravity acting on excess submandibular fat; Isono & Remmers, 1994). The air pressure inside the pharynx differs from atmospheric pressure during airflow and is influenced by the acceleration of flowing gas. This acceleration of gas increases kinetic energy in the narrowed upper airway, and diminishes potential energy (i.e., luminal pressure) in the section of the pharynx below.

Two factors responsible for occlusion of the airway during an apnea are inspiratory flow limitation and closing pressure. Inspiratory flow limitation is the concept that maximal inspiratory flow through the pharynx to the lungs remains relatively constant despite the progressive narrowing of the pharynx and increased resistance to the gas flow. Thus, as resistance against the gas flow increases, more potential energy is lost in overcoming the friction, thus resulting in a loss of luminal pressure and a further reduction of airway patency. Finally, the loss of lateral wall pressure reaches a critical value (i.e., closing pressure) that results in the absence of the pharyngeal lumen (Isono & Remmers, 1994).

In contrast to the passive phase, the active phase of the pharyngeal lumen is stated to occur when muscles that determine the luminal air space are in a state of contraction, and the airway is dilated. A phasic burst of reflexual muscle contraction that dilates the upper airway opposes the collapsing negative pressure produced by the rapid airflow of inspiration (Harper & Sauerland, 1978). The action of upper airway musculature interacts with sources of internal and external pressure, and these muscles are active during wake and sleep. However, for reasons yet to be completely

understood, neuromuscular control of the upper airway is decreased during sleep, and it is hypothesized that changes in neuromuscular input interacts with a variety of factors including neck flexion, positioning of the jaw, and anatomical abnormalities of the upper airway, all of which influence airflow through the pharynx (Isono & Remmers, 1994). For example, when the mouth opens during sleep, the length of the genioglossus and geniohyoid muscles decreases, thus diminishing the effect of efferent neural activity (Isono & Remmers, 1994). Consequently, the ability of muscles which dilate the pharyngeal lumen to compensate for losses of transmural pressure is impaired during sleep by a variety of factors, any of which can contribute to the pathogenesis of OSAS (Isono & Remmers, 1994).

Structural Abnormalities of the Upper Airway in Patients with OSAS

As stated previously, the upper airway is the final common anatomical location for changes in respiratory control and neuromuscular functioning that lead to sleep apnea (Pépin, Levy, Veale, & Ferretti, 1992). Various techniques have been used to examine the upper airway, and OSAS patient samples have been examined during waking and sleep. Lateral radiography cephalometry has revealed that the vast majority of patients with OSAS have abnormalities on cephalometric roentgenograms (Pépin et al., 1992). In a study of 155 patients with OSAS, 150 had at least two abnormalities of the upper airway (Jamieson, Guilleminault, Partinen, & Quera-Salva, 1986). Patients with OSAS have been found to have a longer and thicker soft palate as compared to normal subjects (De Berry-Borowiecki, Kukwa, Blanks, & Irvine, 1988; Lyberg, Krogstad, & Djupesland, 1989). A reduction in the size of the posterior airway space below 5 mm (normal = $11\text{ mm} \pm 1\text{ mm}$) and a value of greater than 24 mm (normal = $15.4\text{ mm} \pm 3\text{ mm}$) for the distance from the mandibular plane to the hyoid bone (anchors

the tongue muscles) has been found to correlate with the A+HI independent of the body mass index (BMI; Partinen, Guilleminault, Quera-Salva, & Jamieson, 1988).

Additionally, cephalometric studies have revealed that the hyoid bone tends to be abnormally low in persons with OSAS (De Berry-Borowiecki, et al., 1988).

Computerized tomography (CT) of the upper airway in a sample of OSAS patients has shown that the cross section area of each level of the pharynx is significantly reduced as compared to normal control subjects (Haponik, et al., 1983). The use of CT has shown increased tongue volume in patients with OSAS (Lowe, Gionhaku, Takeuchi, & Fleetcham, 1986). Using ultrafast CT instead of conventional CT techniques, Shepard, Stanson, Sheedy, and Westbrook (1990) found that the most significant narrowing of the upper airway in OSAS patients during waking occurred at the end of expiration at the level of the oropharyngeal segment. A study of sleeping OSAS patients, found that collapse of the upper airway occurred at pressures ranging from -0.5 to -8.9 cm H₂O (Issa & Sullivan, 1984). Airflow may be altered at various points in the upper airway, and such anatomical defects have a deleterious influence on the airflow mechanics.

Determinants of Daytime Sleepiness and Impaired Functioning in OSAS

Transient arousals from sleep detectable in the EEG as bursts of alpha waves and K-complexes reflect changes in CNS activity. Transient arousals from sleep often occur with the termination of sleep apneas and hypopneas and indicate that an activation of bodily defense mechanisms has occurred in response to a cessation of breathing. However, changes in the EEG associated with the termination of sleep-related respiratory events are not the only alterations in physiological variables that occur in OSAS. A pattern of sympathetic nervous system (SNS) responses generally occurs at the conclusion of an apnea, with increased heart rate being the cardinal feature

(Guilleminault, 1982). Additionally, it is not uncommon for increases in heart rate to reach the point of tachycardia (i.e., over 100 beats per minute in adults; Guilleminault, 1982). Other components of SNS response include increased heart volume, constriction of blood vessels, and deeper and faster respiration (Guilleminault, 1994). At the conclusion of an apnea, respiration is resumed by homeostatic CNS respiratory mechanisms, which maintain blood gases in a range so that the metabolic functions of the body remain normal (Douglas, 1994). Evidence suggests that the hypercapnic ventilatory response to increasing levels of carbon dioxide plays a significant role in arousal from sleep during respiratory changes in OSAS, although hypoxemia, the level of ventilatory effort, and increased inspiratory resistance in the upper airway all make contributions to the respiratory arousal response that resumes breathing (Douglas, 1994). It is arguable that the arousal from sleep which occurs in response to increasing levels of carbon dioxide in OSAS is the most vital defense mechanism in response to breathing abnormalities during sleep, and commonly, arousals to the point of awakening are an important contributor to the decision to seek treatment (Guilleminault, 1982).

The severe sleep disruption experienced by persons with OSAS alters the sleep architecture and diminishes the restorative quality of sleep (Roth, Hartse, Zorick, & Conway, 1980). Sleep fragmentation in the form of numerous, brief arousals from sleep has been demonstrated to be a significant contributor to EDS in the elderly and in persons with certain sleep disorders such as OSAS and periodic limb movement disorder (Carskadon, Brown, & Dement, 1982; Walsh & Lindblom, 1997). Sleep fragmentation is typically manifested by discrete intrusions of alpha wave activity in the sleep EEG, which are referred to clinically as transient arousals from sleep. Transient arousals from sleep often coincide with the termination of apneas and by definition last

from 3 to 14 seconds (ASDA, 1992). In cases of severe OSAS, persons may experience hundreds of these transient EEG changes per night (Guilleminault, 1994). The effect of such severe sleep fragmentation is that the sleep quality of persons with OSAS may be so poor that it approximates total sleep deprivation. Daytime nap studies of patients with OSAS have shown significantly reduced sleep onset latencies compared to those of normal sleepers, with OSAS patients typically falling asleep within 2 to 3 minutes on average during trials of the MSLT (Roth et al., 1982).

Additional evidence for the increased daytime sleepiness which results from sleep fragmentation comes from experimental studies in normal sleepers where approximations of the sleep disruption experienced by persons with OSAS has been shown to significantly reduce sleep onset latencies for the MSLT (Bonnet, 1985, 1986, 1987; MaGee, Harsh, & Badia, 1987; Philip, Stoohs, & Guilleminault, 1994; Roehrs, Merlotti, Petrucelli, Stepanski, & Roth, 1994). Complete awakenings after each minute of sleep in normal subjects have been shown to result in sleep onset latencies during MSLT trials similar to those observed after 64 hours of total sleep loss, even after controlling for the total sleep time (Bonnet, 1986). Changes in the sleep architecture during recovery sleep in persons who have undergone experimental sleep fragmentation are identical to those found in persons who have undergone total sleep deprivation, with rebounds of stages 3 and 4 NREM sleep and REM sleep being observed (Downey & Bonnet, 1987). Additionally, transient EEG arousals occurring as infrequently as once every four minutes during sleep have been shown to result in significantly reduced sleep onset latencies for the MSLT, even when the total sleep time approximated that of the baseline polysomnography (Roehrs et al., 1994). Other forms of sleep disruption including shifts to a lighter stage of sleep have been shown to contribute significantly to

sleepiness as measured by the MSLT (Stepanski, Lamphere, Badia, Zorick, & Roth, 1984). Roth et al. (1980) found that a measure of sleep disruption, which they referred to as arousals subsequent to respiration (which included increases in submental EMG activity and leg movements accompanying respiratory-related arousals), was significantly correlated ($r = -0.67$) with daytime hypersomnolence as measured by the MSLT.

There is strong evidence to suggest that the EDS resulting from sleep fragmentation is the primary cause of impaired performance in persons with OSAS. Bonnet (1985, 1986, 1987) has demonstrated that sleep fragmentation has detrimental effects on daytime cognitive functioning. Subjects who were awakened after each minute of unambiguous sleep (a brief verbal response was required to confirm an awakening) performed significantly worse on a simple reaction time task, completed significantly fewer addition problems correctly in the Wilkinson Addition Task, and performed significantly worse on a symbol substitution task as compared to baseline (Bonnet, 1985). Similar results were obtained in a subsequent experiment where disruptions after each minute of sleep resulted in fewer correct addition problems and performance lapses for a vigilance task (Bonnet, 1986). Hit rates for a vigilance task were significantly impaired in healthy subjects across three levels of sleep disruption on the morning following two consecutive nights of experimental sleep fragmentation (Bonnet, 1987). Lapses in vigilance can often be attributed to repeated microsleeps, during which persons who have experienced significant sleep deprivation involuntarily fall asleep briefly during boring or repetitive tasks (Aldrich, 1994). Furthermore, after 36 consecutive hours of sleep deprivation, the skin conductance orienting response to auditory stimuli, which reflects attentional shifts and allocations of attentional

resources, has been shown to have significantly increased response latency, decreased response amplitude, and faster response habituation which was clearly attributable to sleep loss (McCarthy & Waters, 1997). Thus, physiological changes in the mechanisms underlying attention may also be caused by sleep deprivation and may contribute importantly to impaired performance in patients with OSAS.

Blood gas disturbance in the form of intermittent hypoxemia due to sleep apnea has been shown to be a significant contributor to impaired neuropsychological test performance in persons with OSAS (Bédard, Montplaisir, Malo, Richer, & Rouleau, 1993; Bédard, Montplaisir, Richer, & Malo, 1991; Greenberg, Watson, & Deptula, 1987). Additionally, evidence of memory and spatial skills impairments have been shown to occur in heavy snorers without OSAS, with performance decrements being significantly correlated with the number of oxygen desaturations greater than 4% during sleep (Telakivi et al., 1988). Greenberg et al. (1987) found that OSAS patients performed significantly worse than control subjects on 7 of 14 neuropsychological measures and on a global rating of impairment. Impairments of motor and perceptual-organizational abilities in patients with OSAS were significantly correlated with total time not breathing (i.e., number of apneas multiplied by mean duration) and the lowest blood oxygen desaturation. However, the global rating of impairment was not significantly correlated with either of these measures, but instead was correlated with an estimate of illness duration (Greenberg et al., 1987). Nocturnal hypoxemia from OSAS has also been shown to be an important contributor to impaired executive functioning and vigilance (Bédard et al, 1993; Bédard et al, 1991). The minimum SaO₂ value during sleep was shown to be significantly correlated ($r = -0.76$) with increased reaction time on a vigilance task (Bédard et al, 1991). Although treatment with CPAP over a 6

to 10 month period improved the sleep architecture, respiration during sleep, vigilance, and problems with memory impairment, chronic use of CPAP did not reverse deficits for executive and psychomotor functioning (Bédard et al, 1993). The authors concluded that persistent deficits observed in persons receiving CPAP treatment for moderate to severe OSAS were most likely the result of the severity of nocturnal hypoxemia. It remains to be seen, however, whether nocturnal hypoxemia causes poor quality sleep that impairs functions such as attentional orienting or affects performance via microsleeps.

CPAP Therapy for the Treatment of OSAS

Nasal CPAP was first described as a treatment of OSAS in 1981 (Sullivan, Berthon-Jones, Issa, & Eves, 1981), but was not widely recognized as an acceptable therapy for OSAS until 1985 (Sullivan & Grunstein, 1994). The most important effect of CPAP on the upper airway is that it acts as an air-pressure splint, which maintains the patency of the upper airway by providing continuous pressure to the interior of the airway that prevents it from collapsing (Sullivan & Grunstein, 1994). According to members of the ATS (1994), nasal CPAP is the most effective treatment of OSAS. Nasal CPAP has been demonstrated to be superior to other major modes of treatment for OSAS. In a randomized, controlled trial comparing treatments for OSAS, Lojander et al. (1996) demonstrated CPAP to be superior to palatal surgery and to a conservative management condition (a minimal treatment control condition). In a randomized, crossover study comparing CPAP and an oral appliance (an acrylic polymer mouthpiece) for the treatment of OSAS, CPAP produced no treatment failures as compared to a failure rate of 28% for the oral appliance (Ferguson, Ono, Lowe, Keenan, & Fleetham, 1996).

CPAP uses a facial mask, or some other means of interface (such as intra-nasal tubes) with the upper airway, connected to an air pump that generates pressure in the airway while also allowing the ventilation of expired carbon dioxide. Important therapeutic supplements that can be used with CPAP are oxygen for patients whose SaO₂ levels do not normalize and humidification to make use more comfortable by reducing the tendency for CPAP to cause nasal dryness. The most common method of air pressure delivery is with a nasal mask, which is manufactured in standard sizes, although some companies offer custom fitting for patients with unusual facial contours. Generally, the construction of the nasal masks consists of an outer shell of hard plastic with a soft inner seal constructed of rubber or rubber filled with silicone gel. One major variation of CPAP pressure delivery is nasal prongs, which enter the nostril and form a seal around the opening of the nostril.

The first pressure generating devices for CPAP treatment were air compressors, which although effective, were considered by patients to be bulky and noisy. An advance in the CPAP delivery technology came with the replacement of air compressors with air blowers. Advantages of air blowers include smaller size, greatly reduced noise (they are more easily insulated), and their ability to compensate for a loss of mask pressure due to an imperfect seal with the face (Sanders & Kern, 1992). Currently manufactured CPAP devices are designed to deliver pressures from 2 to 20 cm H₂O, and pressures up to 10 to 12 cm H₂O are usually well tolerated (Sullivan & Grunstein, 1994). Because inhalation and exhalation are conducted through the nasal mask, it is necessary to provide a means for the removal of expired CO₂. Common ways of dealing with this problem are to place a small hole in the mask, or to attach a special

valve to the mask, which serves to deflect expiratory flow out of the system. These devices produce an air leak of approximately 10 to 15 L/min (ATS, 1994).

There are some variations in the pressure delivery of CPAP blower systems. Patients often find exhalation more difficult than inhalation on CPAP because they must exhale against the airflow. The first method of modified pressure delivery designed to address this problem was bi-level positive airway pressure (i.e., BiPAP), which was developed by the Respironics Corporation. The BiPAP system allows the inspiratory and expiratory air pressure levels to be adjusted independently, and was designed to make the positive airway pressure more tolerable. This means of pressure delivery allows for a higher pressure level to be set for inspiration (inspiratory positive airway pressure), without equivalent high pressure for expiration (expiratory positive airway pressure level) and the greater discomfort that this might cause. However, the expiratory positive airway pressure level is essential for treatment efficacy and must be equivalent to the effective pressure level of CPAP, which still requires patients to exhale against the same pressure as would be used with CPAP.

The newest variation on pressure delivery is automatic CPAP, which delivers varying amounts of pressure throughout the night according to the amount required to eliminate snoring and sleep apnea in any given sleep stage and sleep position. A specific range of CPAP pressures is set for the automatic CPAP device, with the lowest pressure being utilized at the beginning of any sleep period. The major theoretical advantage of automatic CPAP is increased patient comfort, because only the amount of pressure that is needed at any given point in the sleep period is delivered. For example, higher pressures are generally required during REM sleep, because of increased irregularity of breathing and atonia, and when persons are sleeping in the supine

position due to the increased collapsibility of the airway specific to this position (Sullivan & Grunstein, 1994). Higher CPAP pressures levels are usually required after the ingestion of even moderate amounts of alcohol or any other muscle relaxant (Sullivan & Grunstein, 1994). The ingestion of alcohol has been shown to increase the number sleep apneas and hypopneas and result in a significant increase in the number of episodes of oxygen desaturation in a sample of men without symptoms of OSAS (Taasan et al., 1981). Alcohol ingestion also has been shown to exacerbate the severity of OSAS (Issa & Sullivan, 1982; Scrima et al., 1982).

There are very few contraindications for the use of nasal CPAP for the treatment of OSAS. Nasal CPAP should not be used with patients who have sustained traumatic head or spinal injury that has caused leakage of cerebrospinal fluid, but otherwise, there are no major contraindications (Sullivan & Grunstein, 1994). When CPAP use is feasible for OSAS, the amount of nasal airway pressure used should restore adequate ventilation and blood oxygenation, and should reduce transient arousals from sleep so that sleep is more continuous and the sleep architecture is normalized (ATS, 1994). The CPAP pressure level is usually titrated in the sleep laboratory setting. Currently, there is no standardized procedure for CPAP titration, but the following are regarded as the primary objectives during a CPAP titration: (1) restoration of a normal respiration pattern; (2) restoration of normal blood oxygen level; (3) normalization of the sleep architecture; (4) elimination of airway resistance (particularly snoring), and; (5) reduction of breathing-related associated transient arousals from sleep to normal levels (ATS, 1994). A range of CPAP pressures is used during the titration procedure with the primary goal being to find a pressure level that is sufficient to prevent apnea, hypopnea, and snoring in all stages of sleep and in all postures of sleep (ATS, 1994; Sullivan &

Grunstein, 1994). Great care must be taken to examine treatment efficacy under these varying conditions because the supine sleep position will require a higher pressure than a lateral or prone sleep position, and REM sleep will require higher CPAP pressures (Sullivan & Grunstein, 1994).

Regular use of CPAP by those with OSAS has been shown to alleviate daytime sleepiness (Frith & Cant, 1985; Kribbs et al., 1993a; Rajagopal, Bennett, Dillard, Tellis, & Tenholder, 1986) and to reduce waking CO₂ levels (Berthon-Jones & Sullivan, 1987). Improvements in cardiovascular functioning such as acute reduction of elevated nocturnal blood pressure levels in OSAS patients with hypertension has been demonstrated with the regular use of CPAP (Fletcher, 1996). Furthermore, chronic application of CPAP for the treatment of OSAS has also been shown to result in a reduction of waking blood pressure levels in OSAS patients (Fletcher, 1996). Additionally, chronic use of CPAP has been shown to be associated with increased waking upper airway dimensions (Ryan, Lowe, Li, & Fleetham, 1991). Long-term assessment of daytime sleepiness with the MSLT has shown continuous improvement of daytime sleepiness in OSAS patients treated with CPAP that was not always evident at the beginning of treatment, with MSLT values continuing to normalize after the first 50 days of treatment (Meurice et al., 1997). Symptoms return quickly when CPAP is discontinued, and only one night's sleep without CPAP has been shown to result in a return to pre-treatment levels of sleepiness and psychomotor vigilance (Kribbs et al., 1993a).

Changes in the Sleep Architecture from CPAP Treatment

The sleep architecture of those suffering from OSAS is significantly different from that of normal sleepers, with OSAS patients spending reduced amounts of time in

SWS and REM sleep and spending increased amounts of time in stage 1 NREM (Roth et al., 1980). The deficit observed for SWS is a clinically significant finding because stages 3 and 4 NREM sleep are particularly restorative (Roth et al., 1980). If a proper level of CPAP pressure is titrated in OSAS patients, there is generally a large rebound of slow wave sleep followed by a similarly large rebound of REM sleep. These rebound phenomena are most dramatic during the first night of CPAP, but rebound of these sleep stages is apparent for about one week after the initiation of treatment (Sullivan & Grunstein, 1994). Regular and proper use of CPAP treatment for OSAS restores the sleep architecture and reduces rates of transient arousals from sleep and awakenings to within normal ranges, provided there is not another comorbid sleep disorder (Isono & Remmers, 1994). For example, Pieters et al. (1996) showed that CPAP treatment in patients with OSAS significantly increased the percentage of stages 3 and 4 NREM sleep, REM sleep, and decreased the number of arousals per hour asleep as compared to baseline.

Surgical Treatment for OSAS

Tracheostomy is an effective surgical intervention for the treatment of OSAS, but is disfiguring and can have serious complications as it is highly prone to opportunistic infections. Uvulopalatopharyngoplasty (UPPP) is a surgical intervention for OSAS in which the uvula and a large portion of the soft palate are excised, and is a better surgical alternative than tracheostomy (Rodenstein, 1992). Surgery is usually performed under general anesthesia and the operation consists of resection of roughly 1.5 cm of the free palate, followed by suturing the posterior and anterior tonsillar pillars together. The anterior and posterior edges of the remaining palate are sutured together to complete the operation. In examinations of the efficacy of UPPP, many investigators

have arbitrarily defined treatment responders as patients who exhibit a reduction of the A+HI by more than 50% from baseline. However, only a minority of patients are classified as UPPP responders and complete elimination of OSAS is rare (Rodenstein, 1992). Several studies have reported success rates of less than 50% (Simmons, Guilleminault, & Miles, 1984; De Berry Borowiecki, Kukwa, & Blanks, 1985). Disregarding treatment responder status, De Berry Borowiecki et al. (1985) reported that 60% of patients had an apnea index of > 20 after UPPP surgery. This is an important result because significantly increased mortality has been noted in patients with an apnea index of >20 per hour (He, Kryger, Zorick, Conway, & Roth, 1988). Complications most commonly occurring after UPPP are transient nasopharyngeal regurgitation, rhinolalia, and infections (Rodenstein, 1992). Mortality may be indirectly increased in patients undergoing UPPP as compared to patients treated with tracheostomy or CPAP. In a 5-year to 8-year follow-up study of patients treated by either UPPP, CPAP, or tracheostomy, all of the patients who died ($n = 14$) were treated by UPPP (He et al., 1988).

Laser-assisted uvulopalatoplasty (LAUP) has been promoted as a safer surgical alternative than the UPPP for the treatment of OSAS (Walker, Grigg-Damberger, & Gopalsami, 1999). Furthermore, research suggests that treatment outcomes from the LAUP procedure are comparable to those obtained with UPPP in terms of reduced A+HI (Mickelson, 1996; Pribitkin et al., 1998). Although the LAUP procedure has been shown to be effective in the treatment of snoring, it has not been shown to be particularly effective in reducing the RDI of OSAS patients, and might only be a viable treatment option for patients with milder illness (Pribitkin et al., 1998).

Non-surgical Treatments for OSAS other than CPAP

Tongue-retaining devices have been tested in large numbers of patients (Lowe, 1994). These devices consist of custom-made mouthpieces that are designed to maintain the patency of the pharynx by creating suction on the tongue and extending it past the teeth. The devices are bulky and tolerance of their use is 50% at best (Lowe, 1994). Additionally, patients must be able to breathe freely through the nose, which may be impossible given the anatomical abnormalities commonly found in patients with OSAS. These devices are generally only effective with patients whose OSAS is ameliorated by sleeping on the side and treatment generally leads to improvement rather than elimination of air resistance (Cartwright & Samelson, 1982). Although weight loss would seem to be a logical recommendation for patients with OSAS, weight loss is rarely ever achieved on a permanent basis and regular use of CPAP is arguably a less demanding and more achievable treatment regimen for OSAS than the maintenance of significant weight loss (Smith, Gold, Meyers, Haponik, & Bleecker, 1985). Finally, positional conditioning can be effective for persons who experience apnea only in the supine sleeping position (Cartwright, 1984; George, Millar, & Kryger, 1988). In this procedure, the patient is trained to avoid sleeping in the supine position, which can be accomplished by wearing a device (e.g., a pajama shirt with a ball sewn into the back) that makes sleeping on the back aversive.

CPAP Compliance

In general CPAP compliance is less than optimal and may even be less than adequate (Kribbs, 1997). Currently, no broadly accepted definition of CPAP compliance in OSAS patients has been formulated, and CPAP compliance has been defined variably across studies (Collard, Pieters, Aubert, Delguste, & Rodenstein,

1997). In general, CPAP compliance has referred to the degree to which patients who accept this form of treatment for OSAS actually use it (Collard et al., 1997). A sample of the various definitions of CPAP compliance that can be found in published research include: self-report estimates of persistent daily use without objective verification of actual time used (Rolfé, Olson, & Saunders, 1991); persistent (although not necessarily daily) use of CPAP at the time of follow-up (Waldhorn et al., 1990; Hoffstein, Viner, Mateika, & Conway, 1992); the average number of hours of daily use as measured from built-in use clocks (Pieters et al., 1996; Fletcher & Lockett, 1991); more than 3 hours per night of daily use (Krieger & Kurtz, 1988); at least 4 hours of use on 70% of the nights measured (Kribbs et al., 1993b); at least 4 hours of use per night on average (Rauscher, Formanek, Popp, & Zwick, 1993); and daily use averaging at least 5 hours per night (Meurice et al., 1994). For persons who use CPAP on nearly a daily basis, compliance has been found to range on average from 5 to 6.5 hours per night (Collard et al., 1997; Krieger & Kurtz, 1988; Fletcher & Lockett, 1991; Engleman et al., 1996). However, most studies using objective means of measuring CPAP use as measured by clocks which record the total time CPAP is on have generally found use averaging less than five hours per night (Berthon-Jones, Lawrence, Sullivan, & Grunstein, 1996; Engleman, Martin, & Douglas, 1994; Rauscher et al., 1993; Reeves-Hoche, Meck, & Zwillich, 1994). Additionally, a close examination of patterns of CPAP use suggests that there can be considerable night-to-night variability of use even in patients considered to be compliant (Kribbs et al., 1993b; Rauscher et al., 1993). For example, a study by Kribbs et al. (1993b) found that only 2 of 35 (6%) patients used CPAP for 7 hours or more per night every night. Furthermore, Rauscher et al. (1993) discovered that only 30% of a sample of patients used CPAP for more than 80% of their reported time

in bed. It is also apparent that patients may take frequent self-prescribed breaks from treatment during situations such as illness, travel, weekends, or may use CPAP on alternate nights or for only part of the night (Rauscher et al, 1993).

The earliest phase of CPAP compliance has been referred to as acceptance (Collard et al., 1997). A considerable barrier to implementing CPAP therapy for OSAS is that many patients refuse CPAP as a treatment option initially or following a full-night or split-night CPAP titration trial in the laboratory (Waldhorn et al., 1990). Rauscher et al. (1993) found that 32% of patients refused a CPAP titration trial from the outset, and 28% of the remaining sample of patients who agreed to undergo a titration trial elected not to use CPAP for treatment of their OSAS. Thus, 49% of the original sample ($N = 95$) refused regular home CPAP treatment for OSAS either initially as a treatment option or after undergoing one night of CPAP titration in the laboratory (Rauscher et al, 1993). Pieters et al. (1996) obtained similar results in a sample of patients with OSAS and found that 24% of patients refused CPAP as a treatment option after a night of titration in the laboratory.

After the initial acceptance of CPAP for home treatment of OSAS, attrition rates are high. In a retrospective study of 168 patients prescribed CPAP therapy, only 64% of patients continued to use CPAP at the time of follow-up (ranged from 1.5 months to 78 months; Rolfe et al., 1991). Similar results for attrition have been obtained in other follow-up studies, with rates of compliance ranging from 65 to 68% (Nino-Murcia, McCann, Bliwise, Guilleminault, & Dement, 1989; Meurice et al., 1994). Although various follow-up intervals have been used within and between studies in attempts to ascertain compliance rates for CPAP, it appears that the first few weeks or, at most, the first few months after CPAP therapy is initiated are critical in promoting long-term

compliance (Nino-Murcia et al., 1989; Collard et al., 1997). Most patients who discontinue CPAP will do so shortly after beginning treatment, and use of CPAP during the first month has been shown to strongly predict use at 3 months (Kribbs et al., 1993b; Fleury, Rakotonanahary, Tehindrazanarivelo, Hausser-Hauw, & Lebeau, 1994). Hoffstein et al. (1992) found that patients who quit CPAP therapy returned their equipment after an average of 3 months. Rolfe et al. (1991) found similar rates for discontinuation of CPAP with 78% of noncompliant patients stopping treatment within two months and 90% by four months. Conversely, for those who continue to use CPAP after the first few months, compliance has been shown to be very stable, with compliant patients continuing to use CPAP for an average of five hours per day over two consecutive follow-up periods covering a total of three years (Pieters et al., 1996).

Methods of Measuring CPAP Compliance

The ATS (1994) recommends routine follow-up to evaluate a patient's response to CPAP therapy, which should include some objective measurement of compliance. In the CPAP treatment literature, methods of measuring compliance have included subjective self-report of daily use and objective methods of measurement including total machine run time, monitored with built-in time clocks, and/or measurement of effective application of CPAP and patterns of use via microchips, which can record the exact amount of time that CPAP is at or near the prescribed pressure (Collard et al., 1997). Microchip technology also allows precise analysis of patterns of use with date and time of each instance that the machine is switched on and off (Collard et al., 1997). Early studies of CPAP compliance in OSAS patients yielded favorable results; however, these investigators did not utilize objective means to verify actual patient compliance with CPAP, with self-report used as the primary measure (e.g., Sanders, Gruendl, & Rogers,

1986). Furthermore, comparisons of patients' estimates and objective measures of compliance reliably show that patients overestimate their average use of CPAP by approximately one hour per night (Rauscher et al., 1993, Kribbs et al., 1993b). The introduction of objective measures of CPAP use has allowed more accurate assessment of compliance. For example, using microchip technology capable of recording machine time on and time off, and the total time at the prescribed pressure for each day, Kribbs et al. (1993a) found that there was often a large variation of use across days for many patients.

Internal clocks by themselves may be adequate for the assessment of CPAP compliance, as consistent and only slight differences between machine time on and time at effective pressure have been found across studies; moreover, there is a very high correlation between these two CPAP use measures, generally on the order of $r = .90$ (Collard et al., 1997). These findings have brought many investigators to the conclusion that monitoring compliance with an internal clock that records total hours of use is necessary and sufficient (Pieters et al., 1996). However, it has been argued that the use of this technique does not allow for an examination of patterns of use, because only the average daily use time is available as opposed to the more detailed night-by-night use data achievable via computer microchip technology (Kribbs, 1997).

Predicting CPAP Compliance

There have been several investigations that have attempted to identify which patient or sleep variables might predict the degree of CPAP compliance. However, no consistent predictive factors have emerged, and statistically significant relations between the variables examined and the criterion of compliance have been shown to be generally weak (Collard et al., 1997). One prospective study of 47 patients with OSAS,

which measured CPAP use covertly with an electronic recording device, found no significant predictors of compliance (Reeves-Hoche et al., 1994). Data analyzed in this investigation included pre-treatment A+HI, age, gender, BMI, education level, prescribed CPAP pressure, and subjective daytime sleepiness (Reeves-Hoche et al., 1994). Employing a similar group of predictor variables, Fletcher and Luckett (1991) also did not find any significant predictors of hourly compliance, although it should be noted that the sample of OSAS patients was very small ($N = 10$). Moreover, initial severity of sleep apnea, effectiveness of CPAP in normalizing the sleep architecture, blood oxygen levels, reported side effects, and prescribed CPAP pressure level were not shown to discriminate between compliant and noncompliant patients (Waldhorn et al., 1990).

In contrast, other studies have found significant relationships between polysomnographic/patient predictor variables and the criterion variables of acceptance and average nightly use of CPAP treatment. Significant correlations have been observed between the average nightly use of CPAP and the polysomnographic parameters of pre-treatment A+HI ($r = .37$), the combined percentage of stage 1 and stage 2 NREM sleep ($r = .30$), and the combined percentage stage 3 and stage 4 NREM ($r = -.31$) sleep during the diagnostic PSG (Meurice et al., 1994). Additionally, Meurice et al. (1994) found significant correlations between nightly use of CPAP and the degree of improvement from baseline to post-treatment for the A+HI ($r = -.34$) and mean SaO₂ during sleep ($r = .38$). Studies of CPAP compliance by Hui et al. (2001) and Bennett, Langford, Stradling, and Davies (1998) have also found significant correlations between the pre-treatment A+HI and CPAP use with correlations of $r = .21$ and $r_s = .34$ being observed respectively. Also,

objective CPAP use has been found to significantly correlate with lowest SaO₂ during the baseline study ($r = -.26$; Rauscher et al., 1993). In a study of 168 patients prescribed CPAP, significantly more patients with both EDS and SaO₂ desaturation below 75% were using CPAP at follow-up (Rolfe et al., 1991). However, it should be noted that this sample included a large number of patients who did not have OSAS and were being treated with CPAP for snoring, and the study did not use objective means of measuring compliance. A significant difference has been observed between patients with milder and more severe OSAS in initial acceptance of CPAP, with only 28% of mild to severe OSAS patients (A+HI between 15 and 30) accepting CPAP therapy versus 95% of those with very severe OSAS (A+HI > 60; Rauscher, Popp, Wanke, & Zwick, 1991b). Hoffstein et al. (1992) found that compliant CPAP users had a significantly higher mean BMI than non-compliant patients. Finally, full-night titration studies are often used to set an effective level of CPAP pressure, but, as a cost saving measure, many sleep disorders centers routinely conduct a 2 to 4 hour baseline and begin CPAP titration for the remainder of the night (i.e., split night studies) if warranted and/or feasible. Fleury et al. (1994) found that acceptance and compliance rates for CPAP in patients who were introduced to therapy during a split-night procedure were comparable to those found for full-night titration studies, concluding that this did not appear to be an important consideration in CPAP compliance. Although Strollo et al. (1996) also did not find a significant difference in the rate of CPAP use between patients who had undergone CPAP titration during a split-night procedure versus a full-night procedure, there was a trend for persons in the full-night condition to use CPAP more on average per night (5.2 vs. 3.8 hours per night); however, the authors caution that the study sample size was small ($n = 24$). Because significant predictors and criteria for compliance have varied

widely among studies, it is likely that long-term compliance with CPAP in OSAS patients is determined by a variety of factors (Pieters et al., 1996).

The variable and weak results that have been obtained in trying to predict CPAP compliance from traditional objective sleep variables have been of limited value in predicting CPAP compliance. Bennett et al. (1998) used the new computerized techniques of digital subtraction of video imagery (to detect subtle arousal-related movements during sleep) and neural network EEG analysis (which examines sleep depth on a continuous scale rather than the traditional sleep staging of Rechtschaffen and Kales [1968] sleep scoring criteria) in an effort to find more sensitive measures of sleep restfulness that might be useful in predicting CPAP compliance. In this mixed sample that included patients with simple snoring through those having severe OSAS, Bennett et al. (1998) found that compliance with CPAP treatment was significantly correlated ($r_s = .39$) with the movement event index, and with two measures of EEG sleep depth (i.e., standard deviation of sleep depth and sleep descent index) derived from the neural network analysis ($r_s = .47$, $r_s = .48$ respectively).

Research also indicates that subjective measures of CPAP satisfaction might predict compliance with CPAP in OSAS patients (Hoffstein et al., 1992). Engleman et al. (1996) specifically focused on subjective indicators of effectiveness and compliance and found that subjectively measured use of CPAP was significantly correlated with pre-CPAP Epworth Sleepiness Scale scores ($r = .22$) and post-CPAP Epworth Sleepiness Scale scores ($r = -.18$). Other important relations between self-reported CPAP use and subjective indicators of improvement were derived from factor analysis (Engleman et al., 1996). CPAP compliance was correlated with positive changes in daytime functioning ($r = .44$), changes in nocturnal symptoms ($r = .35$), and changes in

general functioning/symptoms ($r = .46$). Finally, a “nuisance factor” was associated with reduced compliance ($r = -.15$), and was especially common in those with milder illness (Engleman et al., 1996). In another study, Waldhorn et al. (1990) observed that compliant patients (i.e., using CPAP at the time of follow-up) had significantly more subjective daytime sleepiness as assessed at baseline than noncompliant patients. Rauscher et al. (1991b) found that significantly more OSAS patients who reported EDS accepted CPAP compared to patients who reported little or no EDS. Furthermore, Fletcher and Luckett (1991) found that more severe baseline obstructive sleep apnea symptoms were significantly correlated with perceived improvement after treatment ($r = .62$). Hoffstein et al. (1992) found that significantly more patients who reported no subjective improvement with CPAP discontinued CPAP, which was in contrast to the lower discontinuation rate of patients who reported subjective benefits from CPAP therapy. Finally, Meurice et al. (1994) found that an improvement in a score of subjective daytime sleepiness measured before and after CPAP treatment was significantly correlated ($r = -.40$) with daily use of CPAP.

Side Effects and other Factors in CPAP Noncompliance

Though reports of serious complications for any mode of CPAP are rare, side effects are very common (e.g., skin and eye irritation, nasal dryness and congestion, and rhinorrhea; ATS, 1994). Hoffstein et al. (1992) found that waking up during the night was the most common side effect and was reported by 46% of the sample. For patients who refused CPAP after a titration trial, 15 of 18 patients complained of having trouble falling asleep with the CPAP mask on (Pieters et al., 1996). Lojander et al (1996) found that six of eight persons who refused CPAP after a night of CPAP titration reported that did so because of failure to adjust to the machine. Intolerance of the CPAP mask is a

common complaint, with as many as 43% of patients report discontinuing CPAP therapy for this reason (Rolfe et al., 1991). Pépin, Leger et al. (1992) found that 50% of patients using CPAP complained of at least one side effect due to the nasal mask. Although Waldhorn et al. (1990) found both compliant and noncompliant CPAP patients had a high rate of reported adverse reactions to CPAP (discomfort with the nasal mask was the most common), no significant difference between the groups was demonstrated for any specific complaint. Side effects have been shown to be unrelated to the number of months CPAP was used as assessed at follow-up (Nino-Murcia et al., 1989). Additionally, side effects have been shown to be unrelated to the pressure level prescribed for home use (Pépin, Leger et al., 1992; Rauscher et al., 1993). Side effects have also been shown to be unrelated to self-report of CPAP use (Engleman et al., 1996). Nino-Murcia et al. (1989) found that 50% ($n = 5$) of patients who quit CPAP within the first 2 weeks of initiating therapy did so because of anxiety. Hoffstein et al. (1992) found that 10% of patients complained of claustrophobia from wearing the nasal CPAP mask. Additionally, Kribbs et al. (1993b) found that feelings of claustrophobia when wearing the CPAP mask were reported significantly more often by patients who were irregular users of CPAP (defined as using CPAP ≤ 4 hours per for $\leq 70\%$ of days).

Interventions Aimed at Improving CPAP Compliance

Many of those who accept CPAP for treatment of OSAS at home will discontinue treatment, and such patients generally abandon this mode of therapy in the first three months (Collard et al., 1997; Hoffstein et al., 1992). It has been suggested that the initial period of adjustment to CPAP is the most crucial for compliance, because many OSAS patients who accept CPAP therapy for home treatment quit in as little as 2 weeks (Nino-Murcia et al., 1989). Despite the fact that noncompliance with CPAP

treatment in OSAS patients is a widely recognized problem, surprisingly few interventions have been evaluated for improving CPAP compliance. Support in the form of weekly, followed by monthly telephone contacts was not shown to significantly improve objective compliance for new CPAP users as compared to a control group of chronic CPAP users (Fletcher & Lockett, 1991). Additionally, a study of CPAP use in newly diagnosed OSAS patient by Hui et al. (2000) that also included support in the form of daily, followed by weekly and monthly telephone calls and that had participants meet their physician after the first and second week of CPAP therapy was not shown to significantly improve compliance. Likar, Panciera, Erickson, and Rounds (1997) showed improvement in hourly use of CPAP in patients who participated in one or more (mean = 4) two-hour group education sessions held every six months, with an average increase in nightly use of CPAP from 5.2 hours to 6.3 hours per night. Additionally, education increased nightly use by at least two hours in 10 of 34 patients (29%). Although these results are encouraging, the authors emphasized that there was no control group, and the observed improvement may have been influenced by factors other than the group education sessions (Likar et al., 1997). Chervin, Theut, Bassetti, and Aldrich (1997) compared a group of patients with OSAS who received weekly phone calls to troubleshoot and encourage CPAP use against a group patients who received written information regarding the nature of sleep apnea and the importance of regular use of CPAP. Both interventions increased use as compared to a control group, with average nightly use being 2.7 hours greater in the information group than in the control group and 1.3 hours greater in the phone call group than in the control group, although average nightly use was significantly increased only for the information group.

These authors also found that intervention benefited patients most when it occurred during the first month of beginning CPAP (Chervin et al., 1997).

Habituation of CPAP

Habituation is generally defined as a progressive decrease in the magnitude of a response that occurs with repeated presentations of an eliciting stimulus (Domjan, 1996). Habituation is a basic and common form of learning and is adaptive in the sense that it allows organisms to ignore inconsequential stimuli (Petrinovich, 1984). Furthermore, the habituation of a response represents a relatively permanent form of learning, with observable short-term and long-term effects on behavior (Petrinovich, 1984). The behavioral opposite of habituation is sensitization, which was discovered when existing theories of habituation did not adequately account for all of the changes that might occur in response to repeated presentations of a stimulus (Petrinovich, 1984). Sensitization is generally described as an increase in the magnitude of a response resulting from the repeated presentation of a stimulus that also can have short-term and long-term effects on behavior (Domjam, 1996). According to the dual-process theory, there are separate neural mechanisms or processes for habituation and sensitization that act in opposition to regulate reflex responsivity (i.e., every stimulus is thought to elicit a behavioral response which is the net effect of the competing properties of habituation and sensitization; Groves & Thompson, 1970). Habituation is posited to occur in the stimulus-response (S-R) system, with sensitization thought to occur in the state system (Groves & Thompson, 1970). The S-R system is involved in the direct elicitation of a behavioral response by a stimulus (Groves & Thompson, 1970). The state system reflects the general level of arousal or tendency to respond and may be influenced by stimuli other than the stimulus that originally activated the S-R

system as well as the original stimulus. It is believed that mild to moderate stimulation produces an initial increase in sensitization until a peak level is reached, which is rapidly followed by a decay of the response. In general, sensitization is a transient response, but may be prolonged when a stimulus is very salient or intense. The processes of habituation and sensitization are influenced by the intensity, significance, and timing of stimulus presentations, and both processes are hypothesized to decay with time. Finally, responses to eliciting stimuli that have resulted in habituation are subject to spontaneous recovery and dishabituation (Groves & Thompson, 1970).

Summary

Although there have been an appreciable number of studies that have attempted to determine factors that influence CPAP compliance, inconsistent rather than consistent predictors have been the norm. After ruling out the severity of the condition, positive reinforcement, and economic reasons as explaining factors which influence patient compliance with CPAP, Pieters et al. (1996) suggested that their habituation-like method of allowing the patients to gain experience with the CPAP device and slowly adapt to positive airway pressure for two to four nights in the hospital prior to the titration polysomnography may have resulted in a positive outcome, although this was not tested experimentally. Other authors have emphasized the importance of the manner that CPAP is introduced to the patient in the laboratory, although no scientific evidence or specific information about how this should be accomplished was provided (Sullivan & Grunstein, 1994). Furthermore, Saskin (1997) refers to the importance of allowing patients to acquire experience with the CPAP stimulus with the blower set “on a low pressure”, and states that this procedure is helpful for patients who are uncomfortable with the idea of CPAP, but again, no data are provided. In an

examination of split-night versus full-night CPAP titration, Strollo et al (1996) reported allowing all patients in the study to experience the CPAP stimulus prior to titration, but did not examine this part of their procedure specifically.

Few studies have attempted to improve CPAP compliance with specific interventions. The current study systematically evaluated the potential value of a habituation procedure in promoting adjustment to CPAP in the laboratory, using a well-detailed habituation method, a properly controlled design, and the appropriate statistical analyses. The current study attempted to fill a gap in the CPAP compliance literature by examining the contribution of the manner in which patients were introduced to the unique CPAP treatment stimulus and its influence on the acceptance and use of this treatment for OSAS. Currently, there is no standard practice regarding the best way to introduce patients to CPAP therapy, and methods vary widely across sleep disorders centers. The CPAP habituation procedure consisted of a 30-minute trial during which patients were acclimated to CPAP while experiencing a range of pressures using a standard nasal mask prior to undergoing CPAP titration in the laboratory. Allowing patients to experience CPAP prior to polysomnography with CPAP titration, while they were engaged in typical pre-sleep activities such as watching television or reading, was hypothesized to make the CPAP stimulus less arousing during sleep. Information about CPAP is a valuable resource for patients, but it was hypothesized that in vivo experience with the CPAP apparatus would produce better CPAP compliance post polysomnography. Undoubtedly, many sleep disorders centers provide information about CPAP and allow patients to experience CPAP before treatment; however, there have not been a published scientific study that has specifically examined the role of patients' initial experience with the CPAP stimulus in the laboratory to treatment

outcome. Given the potential health problems associated with untreated OSAS, the efficacy of CPAP treatment for OSAS, and the lack of good treatment alternatives, identifying a method to improve CPAP compliance warranted exploration.

In summary, habituation of CPAP prior to the polysomnography was hypothesized to facilitate the initial adjustment to the treatment, which was hypothesized to be reflected in the sleep architecture during the time of CPAP titration. Improved initial adjustment to the CPAP treatment stimulus was hypothesized increase the acceptance of CPAP and the degree of individual hourly compliance with CPAP therapy for the treatment of OSAS.

Methods

The CPAP habituation study was designed to test the general hypothesis that habituation to the CPAP treatment stimulus prior to attempting to sleep with CPAP would promote the acceptance and use of this form of treatment for OSAS. All participants were scheduled to undergo some form of polysomnography (i.e., split-night or full-night CPAP titration procedure), in a medical sleep disorder center on the night of their participation in the study. The study consisted of two experimental groups and two control groups. Participants in the two experimental groups wore the nasal CPAP mask during wake and experienced a range of air pressures over a 30-minute trial on the evening that their sleep studies were conducted. This occurred prior to the clinical polysomnography. All participants, including those in the two minimal treatment control conditions, received information (see Appendix A) regarding the seriousness of OSAS, the effectiveness of CPAP in treating this condition, and the benefits that they could expect to derive from using CPAP regularly. Participants undergoing the CPAP habituation procedure received this information also because withholding information about such a serious medical problem would not have been in their best interest.

Participants

Of the total of 144 participants that were recruited for the study, 61 participants met inclusion criteria for the data analyses. Participants were patients referred to the Sleep Disorders Center of Alabama (SDA), the HealthSouth Medical Center Sleep Disorders Center (HMC), or the Sleep Disorders Center of the Ochsner Clinic of Baton Rouge (OCBR) for a clinical polysomnography (PSG). The purpose of the clinical PSG was to rule out the presence of OSAS with the possibility of CPAP treatment during the study, or in the case of patients newly diagnosed with OSAS, to undergo a full-night

CPAP titration trial. Participants were approached in the laboratory on the night of their sleep study to determine if they were interested in participating in the experiment. Participants were fully informed about the purpose and nature of the study in accordance with the ethical guidelines of the American Psychological Association (1992) prior to their participation. The participants signed appropriate consent forms (see Appendixes B, C, and D) before any experimental procedure was undertaken. Participation was voluntary, and participants were informed that they could discontinue their participation at any time during the study without penalty. All participants included in the final data set experienced a CPAP titration trial during the course of the clinical PSG on the same night of their participation in the experiment. The experimenter informed the participants of the potential benefits that might result from participating and of the scientific knowledge that might be derived as a result of their participation in the experiment. Participants were followed up after they had been on CPAP treatment at home for an average of approximately seven weeks, at which time objective electronic or time counter data was collected from their CPAP device by a representative of the sleep disorders center.

Although an A+HI of at least five per hour meets the diagnostic criterion for the minimum number of events that is required per hour asleep for OSAS, only participants with an A+HI of at least 10 per hour were included in the study. This inclusion criterion was chosen in light of the current scientific literature, which shows that serious health consequences of OSAS are most likely in those with an A+HI of 10 or more per hour. Any subject deemed eligible to receive CPAP treatment by the referring physician was eligible for either of the experimental conditions. Chart reviews were conducted to screen for presenting complaints of loud and persistent snoring, EDS, and

reports of respiratory pauses during sleep to find appropriate candidates for the study. Participants were not eligible for the study if they have had any previous first hand experience with CPAP or BiPAP. All appropriate candidates were randomly assigned to the experimental or control condition by means of a coin toss. Physicians assigned participants to the split-night or full-night titration condition based upon a clinical evaluation. Because three laboratories were employed to recruit and run participants, an effort was made to maintain a balance between the number of participants participating from each sleep disorder centers. However, extenuating circumstances resulted in relatively fewer participants coming from the OCBR. Men and women were eligible to participate in the study.

Procedure

Four groups of participants were utilized in the experiment. Please refer to Table 1 for a description of the experimental conditions, which will be followed by a

Table 1: Description of the Groups and Relevant Procedures

	Split-Night Titration	Full-Night Titration
Habituation/Education	Group 1: <u>Hab/Ed-SN</u> Standardized Education CPAP Habituation Split-Night CPAP Titration	Group 2: <u>Hab/Ed-FN</u> Standardized Education CPAP Habituation Full-Night CPAP Titration
Education	Group 3: <u>Ed-SN</u> Standardized Education Sleep Disorders Inventory Split-Night CPAP Titration	Group 4: <u>Ed-FN</u> Standardized Education Sleep Disorders Inventory Full-Night CPAP Titration

more detailed explanation of the procedures for each group, in the text. The number of participants for each group varied from the proposed number. The proposed n per group of 23 was not achieved for any of the groups. The number per group in the split-

night conditions approached the proposed number of participants (Hab/Ed-SN = 22, Ed-SN = 21). However, participants for the full-night conditions were scarcer than had been anticipated, and as a result, an *n* of only nine per group was achieved (Hab/Ed-FN = 9, Ed-FN = 9). The proposed number of participants per group for the nightly CPAP use data analysis was derived from a consideration of a nominal alpha level of .05, an acceptable power of .80 (beta = .20, no more than four times alpha), two treatment levels, and an estimated effect size equal to one. The minimum number of participants needed per cell to avoid an excessive chance of a Type II error was derived from Table C.12 of Hinkle, Wiersma, and Jurs (1994). Although 17 participants was the exact number listed, 23 per group were proposed because the CPAP treatment literature suggested that only about 75% of all participants would elect to embark upon CPAP therapy after a undergoing CPAP titration in the sleep laboratory, and that 23 participants per group would be needed to include at least 17 per group for the nightly CPAP use data analysis. The effect size chosen was based on limited information regarding average daily use of CPAP. The average CPAP use in new patients across studies has been shown to be approximately five hours per night with standard deviations of approximately one hour being common. Therefore, in order for this study to have had practical significance, it was predicted that CPAP use should be increased by at least one hour per night on average in those who had undergone CPAP habituation. Further, interventions such as information and education sessions about CPAP have been shown to increase use by at least one hour per night, based on the a review of the CPAP treatment literature.

After they had given their written informed consent to participate, all participants were presented with the written educational briefing contained in Appendix

A regarding obstructive sleep apnea syndrome and its treatment with CPAP. The sleep laboratory directors approved the written CPAP education, and participants received the written educational briefing prior to experiencing any of the other procedures. After the experimenter had presented the consent form and the written information, any questions that the participant had regarding the procedure or the nature of the research were answered at that time. At the conclusion of the educational briefing, participants in the two CPAP habituation groups (Hab/Ed-SN, Hab/Ed-FN) were fitted with a standard nasal CPAP mask and underwent the habituation procedure. The proper mask size was ensured through the use of a measuring device manufactured by the Respiration Corporation. Following this, participants in the CPAP habituation groups placed the nasal CPAP mask (which was attached to the CPAP system with a hose) over his or her nose, and the experimenter affixed the mask with a headgear that consisted of a web of four straps that looped through slots in the CPAP mask and wrapped around the participants head. Next the experimenter informed the participants that the CPAP device was going to be switched on, and they were instructed to breathe normally during CPAP. To ensure that all participants experienced the exact CPAP habituation pressures described, an air leak detection device contained in the CPAP monitoring/control system was used to evaluate the quality of the seal of the mask with the face. If the leak was below a value of 20 milliliters per minute, the mask was considered a good fit, and deemed as delivering the correct pressure. The trial was considered to have begun when the machine was switched on.

Participants in the two Hab/Ed groups experienced a range of CPAP pressures in a systematic fashion while awake. Pressures of 4 cm H₂O through 10 cm H₂O were used. Patients began the trial at 4 cm H₂O, as this was in accordance with the usual

starting CPAP pressure that patients were introduced to during a typical titration procedure. Pressure was increased in increments of 2 cm H₂O every 7.5 minutes until each patient spent 7.5 minutes at 10 cm H₂O, and the CPAP habituation procedure lasted a total of 30 minutes with the mask on and delivering pressure. The final pressure of 10 cm H₂O was selected because a review of the literature revealed that the average prescribed pressure for CPAP across laboratories was approximately 10cm H₂O (e.g., Ferguson et al., 1990; Lojander et al., 1996; Pieters et al., 1996; Waldhorn et al., 1990). The experimenter was present with the participants during the CPAP habituation procedure (with the exception of leaving the room briefly to adjust the CPAP pressure) to monitor for indications that the study should have been discontinued (e.g., panic-like response, severe discomfort), to ensure that participants remained awake, and to confirm that participants were wearing the CPAP mask properly during the procedure. All participants were instructed that they would be informed of each pressure increase before it occurred and that they could request that the pressure be lowered to the previous level if they had difficulty tolerating a pressure increase. The experimenter asked the participants to give a hand signal (a thumbs up) after each pressure increase to indicate that the trial should continue at the new pressure. If participants indicated that they were experiencing discomfort in response to a pressure increase, the pressure was lowered to the previous level, and they were encouraged to keep the mask on for the remainder of the 30-minute trial while continuing at the highest pressure that had been well-tolerated. This was done so that all participants included in the data analysis had an equal number of minutes of experience with CPAP prior to their sleep study. However, the participants were informed that they could discontinue the trial if they remained uncomfortable after returning to the previous

CPAP pressure level. Participants were allowed to engage in any reasonable distracting activity that they chose during the procedure (e.g., watching TV, reading magazines). The experimenter spent approximately 45 to 60 minutes with each patient in the each of the study groups, including the completion of the informed consent. All participants were debriefed informally following the CPAP habituation trial to determine if they had any comments or questions regarding the CPAP habituation procedure.

The CPAP education groups (Ed-SN, Ed-FN) were minimal treatment control groups that did not undergo the habituation procedure. These participants were provided with information regarding the consequences of untreated sleep apnea and the benefits of undertaking CPAP therapy for the treatment of obstructive sleep apnea. In order to account for time spent with the experimenter as a potential confounding variable that could account for a positive outcome in the experiment, the experimenter spent an equal amount of time interacting with the control participants. The control participants spent their time answering questions about their sleep problems with the aid of the Sleep Disorders Inventory (SDI; Waters, unpublished; see Appendix E).

The only different pre-PSG information which the CPAP education control participants were given was that they were told they would be participants in an experiment who would undergo the usual laboratory procedures and would be compared to a second group of participants who would undergo a different procedure to determine if it would help this second group adjust to CPAP better. The CPAP education control participants were also told that the additional information that they provided to questions of the SDI might potentially provide a better understanding of adjustment to CPAP. In accordance with the normal procedures in the participating sleep disorders centers, all persons undergoing a sleep study with the possibility of

CPAP were given the opportunity to get in vivo experience with the CPAP apparatus before undergoing their sleep study. The usual laboratory procedure was to fit persons with a nasal mask if they desired and to use the starting pressure of 4 cm CPAP pressure only. After 10 minutes, the sleep laboratory technician asked these participants if they wished to continue to experience CPAP or if they were satisfied and wished to discontinue. The CPAP education control participants could experience CPAP as much or as little as they desired.

Following their pre-PSG participation, each patient underwent the standard PSG procedures of a split-night or full-night CPAP titration in the sleep disorders centers. Sleep parameter data was collected in accordance with the standard of practice. All participants had electroencephalogram (EEG) electrodes (Grass Instrument silver/silver chloride cup electrodes, 8 mm) placed according to the 10-20 international placement system. Leads were attached for C4, C3, O2, O1, A2, A1, electrooculogram (EOG), and submental electromyogram (EMG). C3 and C4 optimally record sleep spindles and delta waves, and the O1 and O2 placements (located above the occipital regions) are particularly useful in detecting the presence of alpha rhythm and determining sleep onset (Guilleminault, 1982). The EOG placements were referenced to A1 and A2 (neutral sites located on the mastoid process). In addition to scalp and face electrode placements, EMG electrodes were attached to the anterior tibialis muscles of the left and right legs to detect periodic limb movements, which, if they occurred, may have resulted in associated arousals which could have confounded the transient arousal data. Respiration was recorded with thermistors (nasal and oral airflow) and two respiration belts (respiratory effort) to detect the presence of any sleep-related breathing disorders. All sleep periods were scored according to Rechtschaffen and Kales' (1968)

sleep stage scoring criteria. Arousals were scored according to the American Sleep Disorders Association (1992) criteria.

Variables of interest in evaluating the effectiveness of the pre-sleep habituation procedure on sleep quality during CPAP titration included: (1) sleep onset latency (SOL; time elapsed from lights out to 3 consecutive epochs of unambiguous sleep or one epoch of sleep with a K-complex or sleep spindle); (2) percent stage 1 NREM sleep (% 1NREM); (3) percent combined stages 3 and 4 NREM sleep or slow wave sleep (SWS); (4) percent rapid eye movement sleep (REM); (5) number of spontaneous transient arousals per hour asleep (STA); (6) number of awakenings per hour in bed (No. Awake); and (7) sleep efficiency, the percent of time asleep of the total time in bed (SE). CPAP compliance data was recorded by means of a microprocessor units built into the Sullivan Elite model CPAP machine and the Respironics Encore model CPAP machine, or a digital time counter in the case of the Fisher & Paykel model CPAP machine. Participants were made aware that their CPAP machines would record their CPAP usage and that their data would be examined after they had used CPAP for several weeks. Data were downloaded or read from each participant's CPAP machine by home healthcare representatives during the routine follow-up visit or were obtained via mail in the form of electronic data storage cards that the participants removed from their CPAP machines and mailed to the OCBR. The variable of interest for the approximately seven-week follow-up was the mean number of hours CPAP was used each day.

Data Analyses

Analysis #1: Comparison of the demographic characteristics of the sample.

Demographic Variables: (1) pre-treatment A+HI, (2) BMI, and (3) age.

One-way ANOVA was used to compare group means for demographic measures to determine if there were any significant groups differences that might need to be taken into account during the interpretation of the results of the study.

Analysis #2: Effect of CPAP habituation on sleep during the CPAP titration PSG.

Dependent Variables: (1) SOL, (2) % 1NREM, (3) % SWS, (4) % REM, (5) STA, (6) No. Awake, and (7) SE.

The following experimental hypotheses were tested:

1. The SOL during the CPAP titration will be significantly less for participants in the Hab/Ed groups (Hab/Ed-SN, Hab/Ed-FN) as compared to participants in the Ed groups (Ed-SN, Ed-FN).
2. The % 1NREM sleep during CPAP titration will be significantly less in the Hab/Ed groups as compared to the Ed groups.
3. The STA during CPAP titration will be significantly less in the Hab/Ed groups as compared to the Ed groups.
4. The No. Awake during the CPAP titration will be significantly less in the Hab/Ed groups as compared to the Ed groups.
5. The % SWS during CPAP titration will be significantly greater in the Hab/Ed groups as compared to the Ed groups.
6. The % REM during CPAP titration will be significantly greater in the Hab/Ed groups as compared to the Ed groups.
7. Sleep efficiency will be significantly greater during CPAP in the Hab/Ed groups as compared to the Ed groups.

Separate one-way MANOVAs were conducted to compare the treatment versus control group means (Hab/Ed vs. Ed) for the sleep variables (with the exception of

STA) in the split-night and full-night conditions. The Hab/Ed vs. Ed comparisons for split-night and full-night conditions were compared separately because the sleep architecture varies across the night, with the % REM increasing and the % SWS decreasing throughout the night, and split-night participants slept with CPAP during the second half of the night when % REM tends to be higher and % SWS tends to be lower than in the first half of the night. Another important reason for analyzing the split-night and full-night conditions separately was to account for the first-night effect (Agnew, Webb, & Williams, 1966), which are consistent changes in sleep architecture that are attributable to the novel sleeping environment of the laboratory with its recording apparatus. Therefore, the adjustment to sleep in the laboratory would likely be more evident in the sleep architecture of the split-night participants. Pearson's correlation coefficients were computed between the sleep quality and quantity variables to determine if they were significantly correlated for use in MANOVA. Alpha was set at the traditional significance level of $p < .05$ for all tests. It was specified that if a MANOVA were significant, individual one-way ANOVAs (Hab/Ed vs. Ed) would have been conducted for each of the dependent variables to identify specific differences between the group means. The STA was examined separately with t-tests comparing the split-night and full-night conditions separately because data was not available for several participants due to the loss of electronic data at the SDA and the HMC. Analyzing the STA separately allowed a larger n for each of the respective MANOVA tests. Alpha was set at the traditional significance level of $p < .05$.

Analysis #3: Effect of CPAP habituation on the initial acceptance of CPAP for home treatment of OSAS.

Dependent Variable: the proportion of acceptance of CPAP treatment for each group.

The following experimental hypothesis was tested: Acceptance of CPAP will be significantly greater in the Hab/Ed groups as compared to the Ed Groups. Acceptance of CPAP was defined as a participant agreeing to take home a CPAP device with instructions to use on a nightly basis (or during each sleep period) to manage his or her OSAS.

Separate tests of proportions were conducted to examine acceptance of CPAP in the split-night (Hab/Ed-SN vs. Ed-SN) and full-night (Hab/Ed-FN vs. Ed-FN) conditions to determine if severity of OSAS had an influence on acceptance. A chi-square test of independence was used to compare CPAP acceptance in the Hab/Ed-SN versus Ed-SN following polysomnography with CPAP titration in the laboratory. The alpha level was set at $p < .05$ as the criterion to reject the null hypothesis that acceptance of CPAP was independent of group assignment. Fisher's exact test was used to compare acceptance for the Hab/Ed-FN versus Ed-FN due to the small number of participants in these groups. The alpha level was set at $p < .05$ as the criterion to reject the null hypothesis that acceptance of CPAP was independent of group assignment. Because the tests were not significant as predicted, a chi-square test of independence comparing the rates of acceptance of the Hab/Ed groups versus the Ed groups was conducted to examine the overall proportion of CPAP acceptance collapsed across titration conditions.

Analysis #4: Effect of CPAP habituation on the degree of compliance.

Dependent Variable: the mean number of hours per day of CPAP use as measured at approximately a seven-week follow-up.

The following experimental hypothesis was tested: The Hab/Ed-SN and the Hab/Ed-FN groups will have significantly more mean hours of daily CPAP use than the Ed-SN and Ed-FN groups.

A two-way ANCOVA (Hab/Ed vs. Ed x SN vs. FN) test was conducted to compare the groups for the nightly mean number of hours of CPAP use, with the pre-treatment A+HI and gender as covariates. Proposed covariates were the pre-treatment A+HI, age, BMI, and gender, but only pre-treatment A+HI and gender were used as covariates after it was revealed that age and BMI had little bearing on CPAP use (please see the results section). For participants in the full-night titration conditions, the A+HI value was derived from the baseline diagnostic polysomnography. The pre-treatment was proposed as a covariate because persons undergoing split-night CPAP titration are likely to have more severe OSAS than persons undergoing full-night CPAP titration, and one of the principal aims of the study was to test whether CPAP habituation increased CPAP compliance regardless of illness severity. Alpha was set at the traditional significance level of $p < .05$.

Results

The objective of the investigation was to evaluate a CPAP habituation method for introducing OSAS patients to CPAP therapy by determining its effects on sleep during CPAP, and its effects on subsequent acceptance and compliance with CPAP treatment. It was predicted that participants in the CPAP habituation treatment groups would have better sleep quality during the CPAP titration, would be more likely to accept CPAP for a trial of treatment at home, and would be more compliant with treatment in terms of average daily use of CPAP at follow-up, than participants in the CPAP education control groups.

Analysis of the Demographic Data

Data from 61 participants were examined in the analyses, and a total of 48 (78.7%) men and 13 (21.3%) women were included in the final sample. The sample was 91.8% White ($n = 56$) and 8.2% African American ($n = 5$). The majority of the sample (83.6%) was recruited from the two sleep laboratories in Birmingham, Alabama, and approximately an equal number of persons participated from the SDA ($n = 25$) and the HMC ($n = 26$). The remainder of the sample (16.4%) participated at the OCBR ($n = 10$). Means and standard deviations were computed for the complete sample for the variables of age, BMI, and pre-treatment A+HI, which can be found in Table 2. On average, members of the sample were middle-aged, obese, and had severe OSAS.

Table 2. Demographic Characteristics of the Complete Sample ($N = 61$)

Variable	<i>M</i>	(<i>SD</i>)	Range
Age	47.38	(10.49)	26-75
BMI	34.18	(9.29)	19.4 - 76.8
A+HI	45.81	(29.65)	10.0 - 130.7

Split-night CPAP titration was administered to 43 (70.5%) participants, and full-night CPAP titration was administered to the remaining 18 (29.5%) participants by PSG technicians.

Demographic data were examined to determine if there were statistically significant group differences for any of the demographic measures that might need to be taken into account when interpreting the results. One-way ANOVAs, with the Games-Howell multiple comparison test (see below), were used to compare the four groups for statistically significant differences on the demographic variables of age, BMI, and pre-treatment A+HI. Means and standard deviations can be viewed in Table 3.

Table 3. Demographic Comparisons by Group

Variable	Hab/Ed-SN		Ed-SN		Hab/Ed-FN		Ed-FN	
	<i>M</i>	(<i>SD</i>)	<i>M</i>	(<i>SD</i>)	<i>M</i>	(<i>SD</i>)	<i>M</i>	(<i>SD</i>)
Age	49.18	(10.46)	47.19	(11.96)	43.11	(10.51)	47.67	(6.42)
BMI	33.05	(5.14)	37.12	(12.21)	27.71	(6.68)	36.53	(8.96)
A+HI	52.62	(27.76)	54.26	(33.08)	21.60	(10.42)	35.31	(28.37)

A one-way ANOVA that compared Age among the four groups was not significant [$F(3, 57) = 0.71, p = .552$]. The observed power at $\alpha = .05$ for the test was .19. A one-way ANOVA for BMI among the four groups was not significant [$F(3, 57) = 2.67, p = .056$]. The observed power at $\alpha = .05$ for the test was .62. A one-way ANOVA that compared the pre-treatment A+HI among the four groups was significant [$F(3, 57) = 3.97, p = .012$]. A Games-Howell multiple comparison test was used because the homogeneity of variance assumption was untenable for the pre-treatment A+HI, and revealed that participants in the Hab/Ed-FN group had a significantly lower mean pre-treatment A+HI than participants in Hab/Ed-SN ($p < .001$) and Ed-SN ($p = .002$) groups. The observed power for the test at $\alpha = .05$ was .81.

A Chi-square test of homogeneity was conducted to examine if unequal proportions of men and women participated in the split-night and full-night conditions. This revealed a statistically significant difference for the number males and females in the titration conditions $\chi^2 (df = 1, N = 61) = 17.86, p < .001$. Most of the male participants (83.3%) participated in the split-night titration condition, and the most of the female participants (76.9%) participated in the full-night titration condition. A Chi-square test of independence conducted to examine if unequal proportions of men and women participated in the treatment and control conditions was not significant, $\chi^2 (df = 1, N = 61) = 0.06, p = .806$. The results for all proportions are presented in Table 4.

Table 4. Comparison of the Number of Participants per Condition by Gender

Condition	Male	Female	χ^2	df	Significance
Titration					
SN	40	3	17.86	1	$p < .001$
FN	8	10			
Group					
Hab/Ed	24	7	.06	1	$p = .806$
Ed	24	6			

Analysis of the PSG Sleep Variables during the CPAP Titration

Means and standard deviations were calculated for the purpose of comparison of the polysomnographic dependent variables for the habituation treatment and education control participants. The split-night and full night conditions were separated for these comparisons because of well-established differences in the sleep architecture across the night. These can be viewed in Table 5 and Table 6. Again, because of differences in the composition of the sleep architecture across the night, the data for full-night and split-night groups were examined separately. For the purpose of examining the effect of

Table 5. Descriptive Statistics for the Sleep Architecture: Split-Night

Variable	Hab/Ed-SN		<i>n</i>	Ed-SN		<i>n</i>
	<i>M</i>	(<i>SD</i>)		<i>M</i>	(<i>SD</i>)	
SOL	11.77	(17.41)	22	14.31	(13.87)	21
SE	82.90	(10.64)	22	84.21	(10.38)	21
%Stage1	10.17	(6.66)	22	12.52	(12.62)	21
%SWS	10.26	(11.23)	22	7.57	(8.83)	21
%REM	20.95	(11.64)	22	20.40	(13.53)	21
No.Awake	2.96	(1.82)	21	3.37	(1.97)	21
STA	4.74	(5.06)	17	6.89	(8.30)	20

Table 6. Descriptive Statistics for the Sleep Architecture: Full-Night

Variable	Hab/Ed-FN		<i>n</i>	Ed-FN		<i>n</i>
	<i>M</i>	(<i>SD</i>)		<i>M</i>	(<i>SD</i>)	
SOL	8.61	(11.21)	9	13.22	(13.18)	9
SE	87.38	(16.31)	9	80.44	(11.51)	9
%Stage1	4.17	(3.46)	9	9.76	(7.03)	9
%SWS	21.14	(17.95)	9	14.28	(12.24)	9
%REM	16.81	(5.18)	8	14.29	(7.17)	8
No.Awake	1.82	(.92)	8	2.82	(1.68)	9
STA	2.83	(2.71)	7	4.36	(2.35)	5

CPAP habituation on sleep during the CPAP titration, MANOVAs were used to compare the PSG variables between the treatment and control participants (i.e., Hab/Ed-SN vs. Ed-SN; Hab/Ed-FN vs. Ed-FN). Pearson correlation coefficients were computed to examine associations among the PSG sleep variables and are given in Appendixes F and G. The results for one-tailed MANOVAs are contained in Table 7. Neither of the

MANOVA analyses was statistically significant at the $\alpha = .05$ level of significance.

The observed power at $\alpha = .05$ for both MANOVAs was .14. The MANOVAs included all of the PSG sleep variables with the exception of STA, which was not included due to

Table 7. Examination of Habituation versus Education Sleep Architecture during CPAP with MANOVA

Titration	Hotelling's T^2	F	df	P
Split-night	2.48	.36	6,35	.45
Full-night	5.92	.61	6,8	.36

a large number of missing data points, and consequently, was analyzed separately. A one-tailed t-test that compared STA between the Hab/Ed-SN and Ed-SN groups was not significant [$t(35) = 0.93, p = .18$]. The observed power for the test at $\alpha = .05$ was .15. A one-tailed t-test that compared STA between the Hab/Ed-FN and Ed-FN groups was not significant [$t(10) = 1.02, p = .17$]. The observed power for the test at $\alpha = .05$ was .15.

Analysis of the Effect of CPAP Habituation on Acceptance of CPAP

Forty-nine participants (80.3%) accepted CPAP for a trial of treatment at home. The chi-square test of independence and Fisher's exact test (for the full-night comparison) were used to determine if acceptance of CPAP was independent of the group assignment. Acceptance of CPAP was examined separately between the split-night and full-night groups to determine if the severity of OSAS had an influence on acceptance. A chi-square test of independence was not statistically significant, $\chi^2(df = 1, N = 43) = 0.09, p = .767$, for a comparison of the proportion of participants accepting CPAP in the Hab/Ed-SN and Ed-SN groups. Also, Fisher's exact test was not statistically significant ($p = .206$) for a comparison of the proportion of participants

accepting CPAP in the Hab/Ed-FN and Ed-FN groups. The split-night and full-night conditions were collapsed across treatment conditions after it was determined that severity of OSAS did not have significant bearing on acceptance of CPAP, and a Chi-square test of independence computed to evaluate whether unequal proportions of participants accepted CPAP in the habituation treatment and education control conditions. The test was not significant $\chi^2(1, N = 61) = 1.50, p = .221$. Refer to Tables 8, 9, and 10 for exact proportions.

Table 8. Chi-square Test of Independence for CPAP Acceptance: Split-Night

Variable	Treatment	Control	χ^2	df	Significance
CPAP					
Accept	17	17	.09	1	$p = .767$
Reject	5	4			

Table 9. Fisher's Exact Test for CPAP Acceptance: Full-Night

Variable	Treatment	Control	Significance
CPAP			
Accept	6	9	$p = .206$
Reject	3	0	

Table 10. Chi-square Test of Independence for CPAP Acceptance: Complete Sample

Variable	Treatment	Control	χ^2	df	Significance
CPAP					
Accept	23	26	1.50	1	$p = .221$
Reject	8	4			

Analysis of the Effect of CPAP Habituation on the Nightly Use of CPAP

Follow-up data for average nightly CPAP use was available for 44 (89.8%) participants who accepted CPAP for a trial of OSAS treatment at home. Data were

available for 33 men and 11 women. Means and standard deviations were computed for each of the four groups in terms of hours of use per night and can be found in Table 11. Pearson product-moment correlations were computed between proposed potential demographic covariates (age, A+HI, BMI, and gender) and CPAP use to determine if there were significant correlations between the proposed covariates and CPAP use.

Table 11. Mean Hourly Use of CPAP per Night

Group	<i>M</i>	(<i>SD</i>)	Range	<i>n</i>
Hab/Ed-SN	5.07	(2.34)	0.75 – 7.92	15
Ed-SN	5.26	(1.76)	0.37 – 7.47	14
Hab/Ed-FN	2.71	(1.93)	0.67 – 3.78	6
Ed-FN	4.42	(2.07)	1.48 – 8.48	9

Additionally, titration type and CPAP acceptance were included in correlation analyses of variables relevant to the examination of CPAP use in the current study. These correlations can be viewed in Appendix H. There were statistically significant point-biserial correlation coefficients between gender and CPAP use ($r_{pb} = .33, p = .03$), gender and A+HI ($r_{pb} = .36, p = .005$), titration type and A+HI ($r_{pb} = .40, p = .001$), and titration type and CPAP use ($r_{pb} = .32, p = .036$). Men tended to use CPAP more per night than women, and men tended to have a higher pre-treatment A+HI than women. Also, participants in the split-night groups tended to use CPAP more per night than participants in the full-night groups, and participants in the split-night groups tended to have a higher pre-treatment A+HI than participants in the full-night groups. Additionally, there was a statistically significant Pearson phi coefficient between gender and titration type ($\phi = .54, p < .001$), as men tended to be assigned to the split-night condition, and women tended to be assigned to the full-night condition by the clinic

physicians. Finally, a t-test that was computed to assess for a possible gender difference among CPAP use was statistically significant [$t(42) = 2.24, p = .03$] and revealed that men used CPAP ($M = 5.08, SD = 2.00$) an average of 1.61 hours more per night than women ($M = 3.47, SD = 2.24$). The observed power for the test at $\alpha = .05$ was .59.

A two-way ANCOVA (Hab/Ed vs. Ed x SN vs. FN) was used to compare the mean number of hours of daily CPAP use with pre-treatment A+HI and gender as covariates. Gender was included as a covariate because of the observed statistically significant correlation between gender and CPAP use and the statistically significant difference for CPAP use between men and women, and A+HI was included as a second covariate because of the statistically significant difference for A+HI between the Hab/Ed-FN group and the split-night groups. The covariate of baseline A+HI was not significant ($F[1, 38] = 0.08, p = .781$). The observed power for the test at $\alpha = .05$ was .06. The covariate of gender was not significant ($F[1, 38] = 1.86, p = .181$). The observed power for the test at $\alpha = .05$ was .27. There were no statistically significant main effects for treatment ($F[1, 38] = 1.91, p = .175$) or titration type ($F[1, 38] = 1.76, p = .192$). The observed power for the tests of main effects at $\alpha = .05$ was .27 and .25 respectively. The interaction was not significant ($F[1, 38] = 1.45, p = .236$). The observed power for the tests at $\alpha = .05$ was .22.

Discussion

The CPAP habituation procedure plus CPAP education was not shown to be superior to CPAP education alone, the community standard of care for introducing OSAS patients to CPAP therapy. In other words, CPAP habituation did not add anything to CPAP education as a means of increasing CPAP compliance, and no statistically significant differences were observed between participants in the CPAP habituation treatment groups and the education control groups for any of the dependent variables. There were several factors that might have influenced the outcome of the study that will be presented after a discussion of the results.

An examination of the polysomnographic data showed that the sleep architecture of the CPAP habituation treatment and the education control participants was not significantly different for either the split-night or full-night conditions, thus demonstrating no statistically significant advantage to persons who experienced CPAP habituation. Because there were no significant differences for age, BMI, or the A+HI between treatment and control groups in the same titration condition, it does not appear that any of the demographic variables had an influence on the polysomnographic data. Polysomnographic variables that had not been previously examined during split-night CPAP titration, such as awakenings per hour in bed, transient arousals per hour asleep, and sleep onset latency during CPAP, also were not significantly different between participants in the CPAP habituation treatment and education control groups. This is consistent with the CPAP compliance literature, in which only weak correlations have been reported between polysomnographic parameters and various definitions of CPAP compliance. The lack of significant results might provide additional support that polysomnographic parameters have limited linkage with CPAP compliance. Although

there were no statistically significant differences for the PSG variables, the values for sleep quality and sleep quantity variables were in the direction predicted by the hypotheses, with the only exception being that participants in the Ed-SN group had non-significantly higher sleep efficiency than participants in the Hab/Ed-SN group. An inspection of the PSG variables revealed that for most comparisons, the differences were small, and there was a large degree of variability in the data, as the standard deviations for most variables were large, and were larger than the means for many variables in the split-night data. Additionally, the differences were so small that it is unlikely that they would have been clinically meaningful even if they were reliable. However, given that the observed power for the MANOVAs was low (.14), a lack of sufficient statistical power might account for a lack of significant findings, which will be discussed below.

Persons who experienced CPAP habituation were not shown to be more likely to accept CPAP than those who experienced the normal laboratory educational procedures for introducing OSAS patients to CPAP. In an examination of the complete sample, the percentage of persons who accepted CPAP (80.6%) after experiencing CPAP titration in the laboratory was similar to that found in the treatment literature (76%, Pieters et al., 1996; 63%, Strollo et al., 1996; 78%, Fleury et al., 1994; 72%, Rauscher et al., 1993; 83%, Waldhorn et al., 1990). Additionally, although small in number and not statistically significant, twice as many participants who experienced CPAP habituation ($n = 8$) rejected CPAP after a night of titration than participants who experienced education only ($n = 4$). This difference for CPAP acceptance is largely accounted for by the performance of persons in the Hab/Ed-FN group, in which three persons rejected CPAP. In regard to the three individuals who rejected CPAP as a treatment option,

evidence suggests that persons with milder OSAS are less likely to accept CPAP treatment (Rauscher et al., 1991). However, CPAP acceptance in the Hab/Ed-FN group raises the possibility that sensitization to the treatment stimulus might have occurred as opposed to habituation, which will be discussed below.

An examination of nightly use of CPAP after an average follow-up period of 49.1 days did not reveal any advantage for CPAP habituation. The mean of 4.68 (SD = 2.16) hours of CPAP use per night for the complete sample was similar to data for nightly CPAP use in the CPAP compliance literature ($M = 4.7$, Engleman et al., 1994; $M = 4.9$, Rauscher et al., 1993; $M = 4.7$, Reeves-Hoche et al., 1994). Nightly CPAP use of at least four hours was achieved by 61.4% ($n = 27$) of the sample, and use of at least five hours of use per night was achieved by only 56.8 % ($n = 25$) of the sample. Although not statistically significant, split-night participants used CPAP an average of 1.43 hours more per more night than full-night participants, which is a clinically important difference. This difference might be best accounted for by severity of OSAS in terms of the diagnostic A+HI, which has been shown to be significantly correlated with nightly CPAP use (Bennett et al., 1998; Hui et al., 2001; Meurice et al., 1994). Although there was no significant correlation between CPAP use and the baseline A+HI in the current study, there was a significant correlation between titration condition and CPAP use, and assignment of the titration condition was based on severity. Several other correlations were significant. Overall, the pattern of significant correlations suggests that there was an association between gender, OSAS severity, and titration type, with women having less severe OSAS, being more likely to be assigned to full-night CPAP titration, and tending to use CPAP less per night than men. A comparison of CPAP use within the split-night sample revealed that nightly CPAP use was very

similar for the CPAP habituation and education control participants with means of 5.07 (SD = 2.34) and 5.26 (SD = 1.76) hours, respectively. These data were similar to the mean nightly CPAP use of 5.5 hours per night reported in a study by Hui et al. (2000) that offered newly diagnosed OSAS patients extensive support during the first month including daily, followed by weekly supportive telephone calls and that had participants meet with their physician after the first and second week of CPAP use.

An unexpected finding was that women used CPAP significantly less per night than men, with women using CPAP an average of 1.61 less per night. A difference of this magnitude would be clinically meaningful; however, the comparison of men versus women for CPAP use is very similar to the split-night versus full-night comparison for CPAP use, in that the vast majority of the data available for men is from split-night participants ($n = 26$, 78.7%) and, conversely, the vast majority of data available for women is from full-night participants ($n = 8$, 73%). Although women and men did not differ on the variables of age or BMI, there was a highly significant difference ($p = .005$) in OSAS severity, with the men having nearly twice as many respiratory events per hour as the women, with a mean A+HI of 51.25 (SD = 30.65) and 25.73 (SD = 12.64) respectively. Studies by Hui et al. (2001), Bennett et al. (1998), Meurice et al. (1994), suggest that severity of OSAS might best explain the gender difference for CPAP use. Furthermore, there was a statistically significant correlation between gender and A+HI in the current study. Therefore, the difference in CPAP use between men and women might be best accounted for OSAS severity. This is further supported by the fact that there was no statistically significant difference between CPAP use between men and women in the full-night condition with men using CPAP an average of 3.87 (SD = 2.30) hours per night, and women using CPAP an average of

3.61 (SD = 2.19) hours per night (no gender comparison was made for the split-night because there were only three female participants). Additionally, three female participants were using CPAP for 45 minutes or less per day at follow-up, which had a great impact on the mean for CPAP use in women. If these data points were removed, the mean CPAP use of the remaining eight females participants would be 4.50 hours per day, which is very similar to the mean CPAP use for the male sample of 5.08 hours per day. Data from 33 men were compared against data from only 11 women, and if more female participants had been included in the sample, the impact of extreme these scores would have been attenuated.

One explanation for the lack of statistically significant findings for the CPAP habituation procedure was generally low statistical power for the tests. Power was very low for the MANOVAs in particular, with an observed power of only .14 for each analysis. The power of MANOVA is affected by the factors of sample size, effect size, and the degree to which the dependent variables are correlated (Stevens, 1980). The factors of sample size and effect size have the most bearing on the lack of significant findings for the PSG variable data analysis. The sample size achieved in the current study was less than proposed, which was particularly true for the full-night conditions. The number of participants available for data analysis would have been decreased further if STA had been included in the MANOVAs, which is why this PSG variable was analyzed separately. Unfortunately, STA was missing for several participants due to the loss of electronic data by clinic personnel at the SDA and HMC. Increasing the sample size would have improved power for the split-night PSG analyses to a small degree. The addition of participants in the full-night groups might have improved power to a greater degree than the addition of subjects to the split-night groups

considering that the number per group was particularly small. Although an exact post hoc estimate of power for the CPAP acceptance analysis was not calculated, observed power for these tests would likely be comparable to the low power observed for the other analyses (Fleiss, 1981). This is particularly applicable to the acceptance data for the small number of full-night participants. In regard to CPAP use, a large effect size had been estimated for the CPAP habituation procedure. The outcome of the study, particularly when examining the split-night sample, suggests that any positive effect that the CPAP habituation procedure might have produced for CPAP use would be quite small even if it were statistically significant. One of the primary research goals was to not only produce a statistically significant difference in CPAP use with CPAP habituation, but to produce a clinically meaningful improvement of at least one hour of CPAP use per night beyond the level of nightly CPAP use that has been reported without such an intervention.

An examination of factors that might have negatively impacted the research methodology requires consideration when interpreting the results of the study. One problem that was unknown at the beginning of the study was the possibility that participants from the SDA and the HMC would receive a benzodiazepine hypnotic medication during CPAP. The physicians and the clinic support staff informed the patients that if they experienced difficulty sleeping with CPAP, they would receive 5 mg of zolpidem (Ambien), and that the medication could be administered a second time if patients continued to have difficulty sleeping with CPAP. A routine physician order for the technicians to administer 5 mg of zolpidem to patients who were not asleep after one hour on CPAP resulted in a large number ($n = 23$) of potential participants being eliminated from the study. Sixty-one percent ($n = 14$) of participants who received

zolpidem were in the education control groups. This was an uncontrolled source of variance that could not be corrected by the investigator. The use of the hypnotic medication was a serious potential confound, given that the purpose of the study was to evaluate the effectiveness of CPAP habituation in promoting adjustment to and acceptance of CPAP, because it would have offered a competing explanation for adjustment to CPAP for those who received it. More importantly, it is arguable that the participants who received the hypnotic medication would have continued to experience difficulty in adjusting to CPAP without it, which very likely would have been reflected in poor sleep architecture and greater difficulty accepting and using CPAP, i.e., they might have expected to have a more negative experience with the CPAP titration trial because of their tendency to sleep onset and/or sleep maintenance insomnia. As a result, participants who received zolpidem were excluded from the analyses, and thus participants who represented an important segment of the patient population and who might have benefited most from CPAP habituation, could not be included in the current sample. Equally important, the exclusion of participants who received zolpidem might have had a positive impact on the results by artificially inflating the percentage of initial acceptance of CPAP that was observed for the complete sample.

A different segment of the population that was under-represented in the current study was persons aged 65 and older as the vast majority of potential participants in this age group could not be recruited from the SDA and the HMC because Medicare was their primary insurance provider, which required them to receive their CPAP devices at a location other than the SDA (HMC patients also received their CPAP devices at the SDA). Only one participant from the OCBR was included from this age group, and no follow-up data CPAP was available for this individual. This limited the ability to

generalize the findings of the study to persons aged 65 and older, a highly significant population given the tendency for OSAS to occur more frequently and to be more severe in patients as they age. The impact of the virtual exclusion of persons aged 65 and older is likely to be limited in view of a study by Parish, Lyng, and Wisbey (2000), which showed that CPAP use in persons aged 65 and older was very similar to that of persons under age 65.

A methodological problem of the study that should be noted was the use of more than one data collection site, which might have compromised experimental control in terms of the research and clinical procedures. A consequence of utilizing three data collection sites was that different clinic personnel and experimenters interacted with the patient samples at the SDA and the HMC versus the OCBR. Data collection was pursued at the OCBR in response to a five-month delay in beginning the data collection at the SDA, and the unavailability of the Sleep/Wake Disorders Center at the University of Alabama at Birmingham as a data collection site that had been proposed originally. Data collection was relocated from the SDA to the HMC due to a decision by the administration to move the vast majority of potential participants to the HMC for insurance purposes. Because the Birmingham data collection sites were under the same management and used the same clinic personnel, data from the SDA and HMC were combined, and compared with data from the OCBR. Comparisons of the Birmingham vs. OCBR data collection sites between participants from the same group with Mann Whitney U revealed statistically significant differences for SOL ($U = 10.00, p = .026$) and STA ($U = 6.00, p = .023$) for the Hab/Ed-SN participants, and also revealed statistically significant differences for STA ($U = 5.00, p = .007$) and SE ($U = 8.50, p = .018$) for the Ed-SN participants. The observed differences suggest that there might

have been important environmental or procedural differences between the sleep disorders centers. As efforts were made to administer the procedures of the study and the CPAP titration in the same manner to all of the participants, it is possible that idiosyncratic inter-laboratory differences in the scoring of polysomnograms might account for the differences observed in the PSG variables. However, the overall impact of the differences for the PSG variables between participants from the SDA and the HMC versus the OCBR was mitigated by the fact that participants from the OCBR represented a small proportion (16.39%, $n = 10$) of the sample, and that those participants were evenly distributed between the split-night groups (Hab/Ed-SN, $n = 4$; Ed-SN, $n = 4$) and the full-night groups (Hab/Ed-FN, $n = 1$; Ed-FN, $n = 1$) for the PSG data analyses. It is very important to note, however, that there were no statistically significant differences for CPAP acceptance or CPAP use between participants from the Birmingham data collection sites and the OCBR data collection site.

Another methodological consideration was that the CPAP habituation procedure, which consisted of only one 30-minute exposure to different CPAP pressures, might have been inadequate to promote complete habituation to the CPAP stimulus. Edinger and Radtke (1993) demonstrated that a gradual desensitization procedure, which included wearing the CPAP mask with pressure for one hour per day for five consecutive days, allowed an individual who had been unable to tolerate CPAP to be compliant with CPAP as measured at a six-and-one-half-year follow-up. In the context of the Edinger and Radtke (1993) study, the CPAP habituation procedure in the current study consisted of only a single trial and was brief, with only 30 minutes of *in vivo* experience for participants. Although this case report by Edinger and Radtke (1993) addressed desensitization as opposed to habituation, it does suggest that a series

of habituation trials and trials of longer duration might have been beneficial. Such a procedure appears to have merit in particular for patients who experience noteworthy anxiety during CPAP, as anxiety has been shown to be a factor in reduced CPAP compliance (Kribbs et al., 1993b; Nino-Murcia et al., 1989). There also is a remote possibility that sensitization, rather than habituation, might have occurred for participants in the Hab/Ed-FN group. However, the evidence available is contrary to a hypothesis of sensitization in the current study. An examination of the effect of the CPAP habituation procedure showed that only 4 of 69 (5.7%) participants who agreed to undergo the CPAP habituation procedure chose to end the procedure early, and consequently were no longer eligible for the study. An identical number of participants requested to settle on the next to highest CPAP habituation pressure level of 8 cm H₂O as their final habituation setting but were able to complete a 30-minute trial with CPAP. Conversely, 61 of 69 (88.4%) of those who agreed to undergo the CPAP procedure experienced no difficulty tolerating the final pressure setting of 10 cm H₂O, and 65 of 69 participants (94.2%) were able to complete a 30-minute trial of CPAP habituation. However, given that that CPAP habituation did not prove useful in the Hab/Ed-SN group and that any possible benefits from CPAP habituation procedure would be small, it does not appear that it would be beneficial to include more participants in the full-night groups. In fact, if it is the case the CPAP habituation procedure resulted in sensitization or worse outcome in patients with milder OSAS, it would be harmful and unethical to recruit more participants using the procedure as it is currently designed.

Finally, given that acceptance and compliance with CPAP in the current sample of OSAS patients was comparable to what has been observed in the literature, the issue of what degree of compliance is beneficial warrants discussion. It has been established

that OSAS patients regarded as being compliant with CPAP are sleeping without treatment for large parts of the night (Kribbs et al., 1993b, Rauscher et al. 1993; Pieters et al. 1996). This is a very important observation because a study of new CPAP users demonstrated that the residual benefit of CPAP after it was removed following four hours of sleep was very limited, resulting in a mean A+HI of 34.9 for the rest of the night without CPAP (Hers et al., 1997). Similar results were obtained in a study of persistent CPAP users (average treatment duration of 8.43 months) that examined sleep after removal of CPAP following the first four hours of sleep, which again, showed that patients experienced unequivocal sleep apnea (mean A+HI = 28.7) for the remainder to the night (Rauscher, Popp, Wanke, & Zwick, 1991a). However, there also is some evidence to suggest that less than optimal use of CPAP may yield some benefits. A study by Engleman et al. (1994) showed statistically significant improvement in OSAS patients on the MSLT, OSAS symptom ratings, mood, and cognitive performance with objectively monitored CPAP use that averaged only 3.4 hours per night after a four-week follow-up. However, it should be noted that the mean MSLT value of 7.2 minutes to sleep onset remained in a range that is considered indicative of moderate to severe daytime sleepiness, and the other observed improvements were small.

In summary, the CPAP habituation procedure was not demonstrated to add any benefit to CPAP education in an examination of sleep quality during CPAP, acceptance of CPAP, and nightly use of CPAP. There were no significant differences between the CPAP habituation/education treatment groups and education control groups on any of the variables examined. Approximately 80% of the participants agreed to a trial of CPAP treatment at home, which was comparable to initial acceptance rates for CPAP in the treatment compliance literature. The average nightly CPAP use for the complete

sample was 4.68 hours, which was also comparable with average nightly use of CPAP that has been reported in the CPAP compliance literature. Men were found to use CPAP significantly more per night than women, but this difference is most likely accounted for by severity of OSAS as men had twice as many respiratory events per hour than women. There were several factors that might have influenced the results of the study, most notable of which were the exclusion of potential participants who had difficulty adjusting to CPAP and received a hypnotic medication during CPAP, and the exclusion of patients aged 65 and older because of insurance coverage considerations. Additionally, a low statistical power was observed, which limited the ability to detect statistically significant differences.

The goal of the present study was to address a gap in the CPAP compliance literature regarding the importance of the manner in which OSAS patients are introduced to CPAP therapy and its effect on acceptance and compliance. The CPAP habituation procedure was designed in response to problems that had been reported in the CPAP compliance literature including poor sleep with CPAP and the experience of anxiety in some OSAS patients who were non-compliant with CPAP treatment. Future research with CPAP compliance could address a number of issues. Interventions might be tailored to the needs of more specific OSAS patient populations. For persons with milder OSAS, a greater emphasis on education such as that reported by Likar et al., (1997), which includes continuing education throughout treatment and that emphasizes the progressive nature of OSAS might be useful for long-term compliance with CPAP. Replication of the findings of Edinger and Radtke (1993) in a small series of patients would lend much greater support for use of their method of CPAP desensitization in persons who experience significant anxiety during CPAP. Finally, although it appeared

that severity of OSAS accounted for an observed gender difference in nightly CPAP use, research specifically addressing possible gender differences for CPAP compliance is lacking. More women are being diagnosed with OSAS and a recent study by O'Connor, Thornley, and Hanley (2000) found that the ratio of men to women for mild OSAS (classified as an A+HI of 5 to 25) was 2.2 to 1 and was 3.2 to 1 for all OSAS patients in their sample. O'Connor et al. (2000) also noted that 62% of women in their sample experienced the majority of the OSAS in REM sleep as compared to 24% of men. Such a difference suggests that it would be worthwhile to conduct research focusing on possible gender differences for CPAP compliance. Given that OSAS has been shown to be associated with ischemic heart disease (Hung, Whitford, Parsons, & Hillman, 1990) and increased mortality (Partinen, Jamieson, & Guilleminault, 1988) and that CPAP is clearly the treatment of choice for OSAS, the search for means to increase CPAP compliance should be continued.

References

- Agnew, H. W., Jr., Webb, W. B., & Williams, R. L. (1966). The first night effect: An EEG study of sleep. *Psychophysiology*, 2, 263-266.
- Aldrich, M. S. (1994). Cardinal manifestations of sleep disorders. In M. H. Kryger, T. Roth, & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (2nd ed.; pp. 413-425). Philadelphia: W.B. Saunders Company.
- American Psychological Association (1992). Ethical principles of psychologists and code of conduct. *American Psychologist*, 47, 1597-1611.
- American Sleep Disorders Association (1992). EEG arousals: Scoring rules and examples. *Sleep*, 15, 173-184.
- American Sleep Disorders Association (1997). The international classification of sleep disorders: Diagnostic and coding manual (Rev. ed.). Rochester, MN: Davies Printing Company.
- American Thoracic Society (1994). Indications and standards for use of nasal continuous positive airway pressure (CPAP) in sleep apnea syndromes. *American Journal of Respiratory and Critical Care Medicine*, 150, 1738-1745.
- Anch, A. M., Remmers, J. E., & Bunce, H. (1982). Supraglottic airway resistance in normal subjects and patients with occlusive sleep apnea. *Journal of Applied Physiology*, 53, 1158-1163.
- Bédard, M., Montplaisir, J., Malo, J., Richer, F., & Rouleau, I. (1993). Persistent neuropsychological deficits and vigilance impairment in sleep apnea syndrome after treatment with continuous positive airways pressure (CPAP). *Journal of Clinical and Experimental Neuropsychology*, 15, 330-341.
- Bédard, M., Montplaisir, J., Richer, F., & Malo, J. (1991). Nocturnal hypoxemia as a determinant of vigilance impairment in sleep apnea syndrome. *Chest*, 100, 367-370.
- Bennett, L. S., Langford, B. A., Stradling, J. R., & Davies, R. J. O. (1998). Sleep fragmentation indices as predictors of daytime sleepiness and nCPAP response in obstructive sleep apnea. *American Journal of Respiratory and Critical Care Medicine*, 158, 778-786.
- Berthon-Jones, M., & Sullivan, C. E. (1987). Time course of change of ventilatory response to CO₂ with long term CPAP therapy for obstructive sleep apnea. *American Review of Respiratory Disease*, 135, 144-147.
- Berthon-Jones, M., Lawrence, S., Sullivan, C. E., & Grunstein, R. (1996). Nasal continuous positive airway pressure treatment: Current realities and future. *Sleep*, 19, S131-S135.

Bonnet, M.H. (1985). Effect of sleep disruption on sleep, performance, and mood. *Sleep*, 8, 11-19.

Bonnet, M.H. (1986). Performance and sleepiness as a function of frequency and placement of sleep disruption. *Psychophysiology*, 23, 263-271.

Bonnet, M.H. (1987). Sleep restoration as a function of periodic awakening, movement, or electroencephalographic change. *Sleep*, 10, 364-373.

Carskadon, M.A., Brown, E. D., & Dement, W. C. (1982). Sleep fragmentation in the elderly: Relationship to daytime sleep tendency. *Neurobiology of Aging*, 3, 321-327.

Carskadon, M.A., & Dement, W. C. (1977). Sleep tendency: An objective measure of sleep loss. *Sleep Research*, 6, 200.

Cartwright, R. D. (1984). Effects of sleep position on sleep apnea severity. *Sleep*, 7, 110-114.

Cartwright, R. D., & Samelson, C. F. (1982). The effects of a nonsurgical treatment for obstructive sleep apnea syndrome: The tongue-retaining device. *Journal of the American Medical Association*, 248, 705-709.

Chervin, R. D., Theut, S., Bassetti, C., & Aldrich, M. S. (1997). Compliance with nasal CPAP can be improved by simple interventions. *Sleep*, 20, 284-289.

Collard, P., Pieters, T., Aubert, G., Delguste, P., & Rodenstein, D. O. (1997). Compliance with nasal CPAP in obstructive sleep apnea patients. *Sleep Medicine Reviews*, 1, 33-44.

De Berry-Borowiecki, B., Kukwa, A., & Blanks, R. H. I. (1985). Indications for uvulopalatopharyngoplasty surgery for snoring and obstructive sleep apnea. *Archives of Otolaryngology*, 111, 659-663.

De Berry-Borowiecki, B., Kukwa, A., Blanks, R. H. I., & Irvine, C. A. (1988). Cephalometric analysis for diagnosis and treatment of obstructive sleep apnea. *Laryngoscope*, 98, 226-234.

Domjan, M. (1996). *The essentials of conditioning and learning*. Pacific Grove, CA: Brooks/Cole Publishing Company.

Douglas, N. J. (1994). Control of ventilation during sleep. In M. H. Kryger, T. Roth, & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (2nd ed.; pp. 204-211). Philadelphia: W.B. Saunders Company.

Downey, R., & Bonnet, M. H. (1987). Performance during frequent sleep disruption. *Sleep*, 10, 354-363.

Edinger, J. D., & Radtke, R. A. (1993). Use of in vivo desensitization to treat a patient's claustrophobic response to nasal CPAP. *Sleep, 16*, 678-680.

Engleman, H. M., Asgari-Jirhandeh, N., McLeod, A. L., Ramsay, C. F., Deary, I. J., & Douglas, N. J. (1996). Self-reported use of CPAP and benefits of CPAP therapy. *Chest, 109*, 1470-76.

Engleman, H. M., Martin, S. E., & Douglas, N. J. (1994). Compliance with CPAP therapy in patients with the sleep apnoea/hypopnoea syndrome. *Thorax, 49*, 263-266.

Ferguson, K. A., Ono, T., Lowe, A. A., Keenan, S. P., & Fleetham, J. A. (1996). A randomized, crossover study of an oral appliance vs nasal-continuous positive airway pressure in the treatment of mild-moderate obstructive sleep apnea. *Chest, 109*, 1296-1275.

Fleiss, J. L. (1981). *Statistical methods for rates and proportions* (2nd ed). New York: John Wiley & Sons.

Fletcher, E. C. (1996). Can the treatment of sleep apnea syndrome prevent the cardiovascular consequences? *Sleep, 19*, S67-S70.

Fletcher, E., & Luckett, R. A. (1991). The effect of positive reinforcement on hourly compliance in nasal continuous positive airway pressure users with obstructive sleep. *American Review of Respiratory Disease, 143*, 936-941.

Fleury, B., Rakotonanahary, D., Tehindrazanarivelo, A.D., Hausser-Hauw, C., & Lebeau, B. (1994). Long-term compliance to continuous positive airway pressure therapy (nCPAP) set up during a split-night polysomnography. *Sleep, 17*, 512-515.

Frith, R. W., & Cant, B. R. (1985). Severe obstructive sleep apnea treated with long term nasal continuous positive airway pressure. *Thorax, 40*, 45-50.

George, C. F., Millar, T. W., Kryger, M. H. (1988). Sleep apnea and body position during sleep. *Sleep, 11*, 90-99.

Greenberg, G. D., Watson, R. K., & Deptula, D. (1987). Neuropsychological dysfunction in sleep apnea. *Sleep, 10*, 254-262.

Groves, P. M., & Thompson, R. F. (1970). Habituation: A dual-process theory. *Psychological Review, 77*, 419-450.

Guilleminault, C. (1982). Sleep and breathing. In C. Guilleminault (ed.), *Sleeping and waking disorders: Indications and techniques* (pp. 155-182). Menlo Park, CA: Addison-Wesley.

Guilleminault, C. (1994). Clinical features and evaluation of obstructive sleep apnea syndrome. In M. H. Kryger, T. Roth, & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (2nd ed.; pp. 667-677). Philadelphia: W.B. Saunders Company.

Haponik, E. F., Smith, P. L., Bohlman, M. E., Allen, R. P., Goldman, S. M., & Bleecker, E. R. (1983). Computerized tomography in obstructive sleep apnea. *American Review of Respiratory Disease*, 127, 221-226.

Harper, R. M., & Sauerland, E. K. (1978). The role of the tongue in sleep apnea. In C. Guilleminault and W. C. Dement (Eds.), *Sleep Apnea Syndromes* (pp. 219-234). New York: Alan R. Liss.

He, J., Kryger, M. H., Zorick, F. J., Conway, W., & Roth, T. (1988). Mortality and apnea index in obstructive sleep apnea. Experience in 385 male patients. *Chest*, 94, 9-14.

Hers, V., Liistro, G., Dury, M., Collard, P., Aubert, G., & Rodenstein, D. O. (1997). Residual effect of nCPAP applied for part of the night in patients with obstructive sleep apnoea. *European Respiratory Journal*, 10, 973-976.

Hinkle, D.E., Wiersma, W., & Jurs, S.G. (1994). *Applied statistics for the behavioral sciences*. Boston: Houghton Mifflin Company.

Hoffstein, V., Viner, S., Mateika, S., & Conway, J. (1992). Treatment of obstructive sleep apnea with nasal Continuous Positive Airway Pressure. *American Review of Respiratory Disease*, 145, 841-845.

Hui, D. S., Chan, J. K. W., Choy, D. K. L., Ko, F. W. S., Li, T. S. T., Leung, R. C. C., et al. (2000) Effects of augmented continuous positive airway pressure education and support on compliance and outcome in a Chinese population. *Chest*, 117, 1410-1416.

Hui, D. S., Choy, D. K. L., Li, T. S. T., Ko, F. W. S., Wong, K. K., Chan, J. K. W., et al. (2001). Determinants of continuous positive airway pressure compliance in a group of Chinese patients with obstructive sleep apnea. *Chest*, 120, 170-176.

Hung, J., Whitford, E.G., Parsons, R. W., & Hillman, D. R. (1990). Association of sleep apnoea with myocardial infarction in men. *Lancet*, 336, 261-264.

Issa, F. G., & Sullivan, C. E. (1982). Alcohol, snoring, and sleep apnea. *Journal of Neurology, Neurosurgery, and Psychiatry*, 45, 353-359.

Issa, F. G., & Sullivan, C. E. (1984). Upper airway closing pressures in obstructive apnea. *Journal of Applied Physiology*, 57, 520-527.

Isono, S., & Remmers, J. E. (1994). Anatomy and physiology of upper airway obstruction. In M. H. Kryger, T. Roth, & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (2nd ed.; pp. 642-656). Philadelphia: W.B. Saunders Company.

Jamieson, A., Guilleminault, C., Partinen, M., & Quera-Salva, M. A. (1986). Obstructive sleep apneic patients have craniomandibular abnormalities. *Sleep*, 9, 469-477.

Kribbs, N.B. (1997). Methods and problems of treatment compliance in obstructive sleep apnea. In M. R. Pressman & W. C. Orr (Eds.), *Understanding sleep: The evaluation and treatment of sleep disorders* (pp. 299-313). Washington, DC: American Psychological Association.

Kribbs, N.B., Pack, A. I., Kline, L.R., Getsy, J.E., Schuett, J.S., Henry, J.N., et al. (1993a). Effects of one night without nasal CPAP treatment on sleep and sleepiness in patients with obstructive sleep apnea. *American Review of Respiratory Disease*, 147, 1162-1168.

Kribbs, N.B., Pack, A. I., Kline, L. R., Smith, P. L., Schwartz, A. R., Schubert, N. M., et al. (1993b). Objective measurement of patterns of nasal CPAP use by patients with obstructive sleep apnea. *American Review of Respiratory Disorders*, 147, 887-895.

Krieger, J., & Kurtz, D. (1988). Objective measurement of compliance with nasal CPAP treatment for obstructive sleep apnoea syndrome. *European Respiratory Journal*, 1, 436-438.

Kripke, D. F., & Ancoli-Israel, S. (1983). Epidemiology of sleep apnea among the aged: Is sleep apnea a fatal disorder? In C. Guilleminault & E. Lugaresi (Eds.), *Sleep/Wake Disorders: Natural History, Epidemiology, and Long-term Evolution* (pp. 137-142). New York: Raven Press.

Likar, L. L., Panciera, T. M., Erickson, A. D., & Rounds, S. (1997). Group education sessions and compliance with nasal CPAP therapy. *Chest*, 111, 1273-1277.

Lojander, J., Maasilta, P., Partinen, M., Brander, P. E., Salmi, T., & Lehtonen, H. (1996). Nasal-CPAP, surgery, and conservative management for treatment of obstructive sleep apnea syndrome. *Chest*, 110, 114-119.

Lowe, A. A (1994). Dental appliances for the treatment of snoring and obstructive sleep apnea. In M. H. Kryger, T. Roth, & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (2nd ed.; pp. 722-735). Philadelphia: W.B. Saunders Company.

Lowe, A. A., Gionhaku, N., Takeuchi, K., & Fleetcham, J. A. (1986). Three-dimensional reconstructions of tongue and airway in adult subjects with obstructive sleep apnea. *American Journal of Orthodontics and Dentofacial Orthopedics*, 90, 364-374.

Lyberg, T., Krogstad, O., & Djupesland, G. (1989). Cephalometric analysis in patients with obstructive sleep apnea syndrome. II. Soft tissue morphology. *Journal of Laryngology and Otolaryngology*, 103, 293-297.

Magee, J., Harsh, J., & Badia, P. (1987). Effects of experimentally-induced sleep fragmentation on sleep and sleepiness. *Psychophysiology*, 24, 528-534.

McCarthy, M. E., & Waters, W. F. (1997). Decreased attentional responsivity during sleep deprivation: Orienting response latency, amplitude, and habituation. *Sleep*, 20, 115-123.

Meurice, J., Dore, P., Paquereau, J., Neau, J., Ingrand, P., Chavagnat, J., et al. (1994). Predictive factors of long-term compliance with nasal continuous positive airway pressure treatment in sleep apnea syndrome. *Chest*, 105, 429-433.

Meurice, J. C., Paquereau, J., Neau, J. P., Caron, F., Dore, P., Ingrand, P., et al. (1997). Long-term evolution of daytime somnolence in patients with sleep apnea/hypopnea syndrome treated by continuous positive airway pressure. *Sleep*, 20, 1162-1166.

Mickelson, S. A. (1996). Laser-assisted uvulopalatoplasty for obstructive sleep apnea. *Laryngoscope*, 106, 10-13.

Nino-Murcia, G., McCann, C. C., Bliwise, D. L., Guilleminault, C., & Dement, W. C. (1989). Compliance and side effects in sleep apnea patients treated with nasal continuous positive airway pressure. *Western Journal of Medicine*, 150, 165-169.

O'Connor, C., Thornley, K.S., & Hanly, P. J. (2000). Gender differences in polysomnographic features of obstructive sleep apnea. *American Journal of Respiratory and Critical Care Medicine*, 161, 1465-1472.

Orr, W. C. (1997). Obstructive sleep apnea: Natural history and varieties of the clinical presentation. In M. R. Pressman & W. C. Orr (Eds.), *Understanding sleep: The evaluation and treatment of sleep disorders* (pp. 267-281). Washington, DC: American Psychological Association.

Parish, J. M., Lyng, P. J., & Wisbey, J. (2000). Compliance with CPAP in elderly patients with OSA. *Sleep Medicine*, 1, 209-214.

Partinen, M. (1994). Epidemiology of sleep disorders. In M. H. Kryger, T. Roth, & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (2nd ed.; pp. 437-452). Philadelphia: W.B. Saunders Company.

Partinen, M., Guilleminault, C., Quera-Salva, M.A., & Jamieson, A. (1988). Obstructive sleep apnea and cephalometric roentgenograms. *Chest*, 93, 1199-1205.

Partinen, M., Jamieson, A., & Guilleminault, C. (1988). Long-term outcome for obstructive sleep apnea syndrome patients. *Chest*, 94, 1200-1204.

Pépin, J. L., Ferretti, G., Veale, D., Romand, P., Coulomb, M., Brambilla, C., et al. (1992). Somnofluoroscopy, computerized tomography and cephalometry in the assessment of the airway in obstructive sleep apnoea. *Thorax*, 47, 150-6.

Pépin, J. L., Lévy, P., Veale, D. & Ferretti, G. (1992). Evaluation of the upper airway in sleep apnea syndrome. *Sleep* 15, S50-S55.

Petrinovich, L. (1984). A two-factor dual-process theory of habituation and sensitization. In H. V. S. Peeke & L. Petrinovich (Eds.), *Habituation, sensitization, and behavior* (pp. 17-55). Orlando, FL: Academic Press, Inc.

Philip, P., Stoohs, R., & Guilleminault, C. (1994). Sleep fragmentation in normals: A model for sleepiness associated with upper airway resistance syndrome. *Sleep*, 17, 242-247.

Pieters, T., Collard, P., Aubert, G., Dury, M., Delguste, P., & Rodenstein, D. O. (1996). Acceptance and long-term compliance with nCPAP in patients with obstructive sleep apnoea syndrome. *European Respiratory Journal*, 9, 939-944.

Pribitkin, E. A., Schutte, S. L., Keane, W. M., Mao, V., Cater, J. R., Doghramji, K., et al. (1998). Efficacy of laser-assisted uvulopalatoplasty in obstructive sleep apnea. *Otolaryngology: Head and Neck Surgery*, 119, 643-647.

Rajagopal, K.R., Bennett, L.L., Dillard, T.A., Tellis, C. J., & Tenholder, M. F. (1986). Overnight nasal CPAP improves hypersomnolence in sleep apnea. *Chest*, 90, 172-176.

Rauscher, H., Formanek, D., Popp, W., & Zwick, H. (1993). Self-reported vs measured compliance with nasal CPAP for obstructive sleep apnea. *Chest*, 103, 1675-1680.

Rauscher, H., Popp, W., Wanke, T., & Zwick, H. (1991a). Breathing during sleep in patients treated for obstructive sleep apnea: Nasal CPAP for only part of the night. *Chest*, 100, 156-159.

Rauscher, H., Popp, W., Wanke, T., & Zwick, H. (1991b). Acceptance of CPAP for sleep apnea. *Chest*, 100, 1019-1023.

Rechtschaffen, A., & Kales, A. (1968). *A manual of standardized terminology, techniques, and scoring system for sleep stages of human subjects*. Publication No. 204. Washington, DC: National Institutes of Health.

Reeves-Hoche, M. K., Meck, R., & Zwillich, C. W. (1994). Nasal CPAP: An objective evaluation of patient compliance. *American Journal of Respiratory and Critical Care Medicine*, 149, 149-154.

Rodenstein, D. O. (1992). Assessment of uvulopalatopharyngoplasty for the treatment of sleep apnea syndrome. *Sleep*, 15, S56-S62.

Roehrs, T., Merlotti, L., Petrucelli, N., Stepanski, E., & Roth, T. (1994). Experimental sleep fragmentation. *Sleep*, 17, 438-443.

Roehrs, T., Zorick, F., Wittig, R., Conway, W., & Roth, T. (1989). Predictors of objective level of daytime sleepiness in patients with sleep-related breathing disorders. *Chest*, 95, 1202-1206.

Rolfe, I., Olson, L. G., & Saunders, N. A. (1991). Long-term acceptance of continuous positive airway pressure in obstructive sleep apnea. *American Review of Respiratory Disease*, 144, 1130-1133.

Roth, T., Hartse, K. M., Zorick, F., & Conway, W. (1980). Multiple naps and the evaluation of daytime sleepiness in patients with upper airway sleep apnea. *Sleep*, 3, 425-439.

Roth, T., Roehrs, T., & Zorick, F. (1982). Sleepiness: Its measurement and determinants. *Sleep*, 5, S128-S134.

Ryan, C. F., Lowe, A. A., Li, D., & Fleetham, J. A. (1991). Magnetic resonance imaging of the upper airway in obstructive sleep apnea before and after chronic nasal continuous positive airway pressure therapy. *American Review of Respiratory Disease*, 144, 939-944.

Sanders, M. H. (1994). Medical therapy for sleep apnea. In M. H. Kryger, T. Roth, & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (2nd ed.; pp. 678-693). Philadelphia: W.B. Saunders Company.

Sanders, M. H., Gruendl, C. A., & Rogers, R. M. (1986). Patient compliance with nasal CPAP therapy for sleep apnea. *Chest*, 90, 330-333.

Sanders, M. H., & Kern, N. (1992). Obstructive sleep apnea treated by independently adjusted inspiratory and expiratory positive airway pressures via nasal mask: Physiologic and clinical implications. *Chest*, 98, 317-324.

Saskin, P. (1997). Obstructive sleep apnea: Treatment options, efficacy, and effects. In M. R. Pressman & W. C. Orr (Eds.), *Understanding sleep: The evaluation and treatment of sleep disorders* (pp. 283-297). Washington, DC: American Psychological Association.

Scrima, L., Broudy, M., Nay, K., & Cohn, M. A. (1982). Increased severity of obstructive sleep apnea after bedtime alcohol ingestion: Diagnostic potential and proposed mechanism of actions. *Sleep*, 5, 318-328.

Shepard, J.W. (1994). Cardiorespiratory changes in obstructive sleep apnea. In M. H. Kryger, T. Roth, & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (2nd ed.; pp. 657-666). Philadelphia: W.B. Saunders Company.

Shepard, J.W., Stanson, A. W., Sheedy, P. F., & Westbrook, P. R. (1990). In F. G. Issa, P. M. Suratt, & J. E. Remmers (Eds.), *Sleep and respiration* (pp. 273-282). New York: Wiley-Liss.

Simmons, F. B., Guilleminault, C., & Miles, L. (1984). The palatopharyngoplasty operation for snoring and sleep apnea: An interim report. *Otolaryngology: Head, and Neck Surgery*, 92, 375-380.

Smith, P. L., Gold, A. R., Meyers, D. A., Haponik, E. F., & Bleecker, E. R. (1985). Weight loss in mildly to moderately obese patients with obstructive sleep apnea. *Annals of Internal Medicine*, 103, 850-855.

Stepanski, E., Lamphere, J., Badia, P., Zorick, F., & Roth, T. (1984). Sleep fragmentation and daytime sleepiness. *Sleep*, 7, 18-26.

Stevens, J.P. (1980). Power of the multivariate analysis of variance tests. *Psychological Bulletin*, 88, 728-737.

Strollo, P. J., Sanders, M. H., Costantino, J. P., Walsh, S. K., Stiller, R. A., & Atwood, C. W. (1996). Split-night studies for the diagnosis and treatment of sleep-disordered breathing. *Sleep*, 19, S255-S259.

Sullivan, C. E., Berthon-Jones, M., Issa, F. G., & Eves, L. (1981). Reversal of obstructive sleep apnea by continuous positive airway pressure applied through the nares. *Lancet*, 1, 862-865.

Sullivan, C. E., & Grunstein, R. R. (1994). Continuous positive airway pressure in sleep-disordered breathing. In M. H. Kryger, T. Roth, & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (2nd ed.; pp. 694-705). Philadelphia: W.B. Saunders Company.

Taasan, V., Block, A. J., Boysen, Wynne, J. W., White, C., & Lindsey, S. (1981). Alcohol increases sleep apnea and oxygen desaturation in asymptomatic men. *American Journal of Medicine*, 71, 240-245.

Telakivi, T., Kajaste, S., Partinen, M., Koskenvuo, M., Salmi, T., & Kaprio, J. (1988). Cognitive function in middle-aged snorers and controls: Role of excessive daytime somnolence and sleep-related hypoxic events. *Sleep*, 11, 454-462.

Waldhorn, R. E., Herrick, T. W., Nguyen, M. C., O'Donnell, A. E., Soderro, J., & Potolicchio, S. J. (1990). Long-term compliance with nasal continuous positive airway pressure therapy of obstructive sleep apnea. *Chest*, 97, 33-38.

Walker, R. P., Grigg-Damberger, M. M., & Gopalsami, C. (1999). Laser-assisted uvulopalatoplasty for the treatment of mild, moderate, and severe obstructive sleep apnea. *Laryngoscope*, 109, 79-85.

Walsh, J. K., & Lindblom, S. S. (1997). Psychophysiology of sleep deprivation and disruption. In M. R. Pressman & W. C. Orr (Eds.), *Understanding sleep: The evaluation and treatment of sleep disorders* (pp. 73-110). Washington, DC: American Psychological Association.

White, D. P. (1994). Central sleep apnea. In M. H. Kryger, T. Roth, & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (2nd ed.; pp. 630-641). Philadelphia: W.B. Saunders Company.

Young, T., Palta, M., Dempsey, J., Skatrud, J., Weber, S., & Badr, S. (1993). Occurrence of sleep disordered breathing among middle-aged adults. *New England Journal of Medicine*, 328, 1230-1235.

Appendix A

Standardized CPAP Education

What is nasal continuous positive airway pressure and how does it work?

Nasal continuous positive airway pressure or CPAP delivers a continuous stream of pressured air through your nose that prevents your airway from collapsing during sleep. It is a safe and extremely effective treatment for sleep apnea, which has been in use for almost 20 years.

Health benefits of CPAP therapy for sleep apnea syndrome.

There are several important reasons that guided your doctor to order a sleep study with CPAP for you. An immediate positive consequence of using CPAP is that many persons notice a significant decrease in sleepiness, which is accompanied by increased feelings of energy and wellbeing. A more long-term, and possibly more important consequence of using CPAP is decreased risk for serious medical problems, which might be caused by untreated sleep apnea. Such problems include hypertension, heart and lung problems, and stroke. Persons with untreated sleep apnea have also been shown to have a higher incidence of serious automobile and work accidents resulting from irresistible urges to sleep. Even if you do not experience excessive sleepiness, the regular use of CPAP every time you sleep for the whole time you are asleep is very important in protecting you from the other problems that untreated sleep apnea might cause. Finally, research has shown that even one night of sleep without CPAP results in a return to how persons with sleep apnea were feeling and functioning before they began using CPAP to treat their sleep apnea.

What to do if there are problems with your CPAP therapy.

CPAP therapy works best when it is used every time you sleep for the entire time that you are in bed. If you experience any problems with CPAP, which are causing you to not use your machine or to use it less than all night, every night, call your home healthcare company representative at (205) 599-1020 for Alabama residents or (225) 767-1403 for Louisiana residents. Many problems with CPAP can be eliminated or reduced.

Appendix B

Consent Form for Participation in CPAP Adaptation, Form A

EXPERIMENTAL GROUP

1. **Study Title:** Examination of a Method for Increasing Adjustment to and Compliance with Nasal Continuous Positive Airway Pressure (CPAP) in Persons with Obstructive Sleep Apnea Syndrome.
2. **Performance Sites:** Sleep Disorders Center of Alabama, Inc. and HealthSouth Medical Center Sleep Disorders Center
3. **Investigators:**
Principal Investigator: Jack A. Johnson, Jr., MA. Mr. Johnson can be reached at (205) 982-9437 from 5:30 p.m. to 7:30 p.m.

Sub-Investigator: G. Vernon Pegram, Ph.D. Dr. Pegram can be reached at (205) 599-1020 or (205) 934-7110 from 8 a.m. to 5 p.m.
4. **Purpose of the Study:** To examine a method that may help patients to adjust better to nasal Continuous Positive Airway Pressure (called CPAP) treatment for sleep apnea.
5. **Subject Inclusion:** Any person age 19 or older who has been referred by his or her physician for a sleep study with CPAP (or with CPAP if indicated) is eligible for participation in this study. Only eligible persons have been invited to participate. Persons who have had a severe head or spinal injury that resulted in leakage of cerebrospinal fluid (the fluid that surrounds and cushions the brain) are not eligible to participate.
6. **Number of Subjects:** 92 Total People will participate in the experiment.
7. **Study Procedures:** All research subjects will be spending the night in the sleep laboratory for a diagnostic evaluation, which might include a CPAP trial aimed at finding a proper level of treatment (air pressure) for obstructive sleep apnea, a routine procedure for persons who have this sleep disorder. Some persons may not receive a CPAP trial during the course of the sleep study if they do not have obstructive sleep apnea or if the degree of their illness does not clinically require CPAP treatment. The diagnostic sleep study will occur immediately following each subject's participation in the research study. CPAP delivers a constant stream of pressurized air through your nose, which prevents you from stopping breathing during sleep. There will be no change in the way that CPAP is normally used in the Sleep

Disorders Center of Alabama, Inc. or the HealthSouth Medical Center Sleep Disorders Center (depending on which sleep laboratory you are having your examination) with the only difference being that you will wear the CPAP mask with air pressure before bedtime while you are not in bed and not attempting to fall asleep. The total time that you will wear the CPAP mask with pressure is approximately 30 minutes, during which time you will experience a range of gradually building CPAP pressures that is commonly experienced by persons during the course of a sleep study with CPAP. You will be started on the lowest pressure setting that is normally used, and the pressure will be increased in steps until it reaches the average pressure setting that is most commonly required to prevent sleep apnea. You will experience each of the four pressure settings for exactly seven and a half minutes each. The pressure will be increased every seven and a half minutes and will increase a total of three times before the experiment is finished. During the time you are wearing the CPAP mask, you may engage in any relaxing activity that you choose including watching television or reading. Data from your sleep study and home CPAP use (if you are prescribed CPAP) will be compared against persons receiving the routine laboratory procedures. All persons will receive written information. Your participation will not reduce or modify information that is normally gathered during the course of a sleep study. Finally, the proposed study is not an experimental procedure, but rather is a variation of the CPAP procedure, which has been designed, with the aim of making adjustment to CPAP easier.

8. **Benefits:** This study may benefit you in your adjustment to CPAP and will further scientific knowledge in the field of sleep medicine concerning the treatment of obstructive sleep apnea syndrome.
9. **Risks/Discomforts:** One risk is that some persons report experiencing anxiety, which may take the form claustrophobia, when wearing the CPAP mask. In general, there is only one major reason that CPAP should not be used for the treatment of sleep apnea. CPAP should not be used with persons who have suffered a very severe head or spinal injury that caused leakage of cerebrospinal fluid (the fluid which surrounds and cushions the brain). As mentioned above, persons who have had an injury of this nature are not eligible to participate.
10. **Right to Refuse:** You have the right to end your participation in this research study at any point that you choose and for any reason that you might have without penalty to yourself. Choosing to drop out of the study will not affect the treatment that you will receive during

your sleep study tonight or at any other time regarding your sleep apnea.

11. Privacy:

The results of this study may be published in a research paper, but your identity or the fact that you have participated in this study will never be made available to public or any other agency.

Your participation in this research study will be kept confidential. However, during the study, representatives from the US Food and Drug Administration (FDA) will be allowed to review your medical records that relate to the study. The Baptist Health System Human Research Review Board may also review these records. The records will identify you only by your initials and an assigned research study number, not by your name.

When you sign the consent document, you are giving your doctor permission to release your research study medical records for review. The representatives described in the paragraph above will use these records only to carry out their obligations associated with this study. Every effort will be made to ensure that your study records are not used for any other purpose, or given to anyone else, except when required by law.

12. Financial Information:

You will not be paid to participate in this study, and there will be no cost to you for participating.

The Baptist Health System Inc, Sleep Disorder Center of Alabama, and Mr. Jack Johnson have made no provision for monetary compensation in the event of physical illness and/or injury resulting from this research study. In the event of such injury, medical treatment is available but is not free of charge. There is no monetary (or other) form of compensation for my participation in this study.

If you have any questions about compensation or medical treatment for research-related injuries, contact G. Vernon Pegram, Ph.D. or Mr. Jack Johnson at the Sleep Disorder Center of Alabama at 599-1020.

13. Alternatives:

Currently, CPAP is the best treatment for sleep apnea for most persons. Your doctor is available to discuss other treatment options with you. The current study is not a treatment, but rather an examination of a way of helping people adjust to a treatment that they might be prescribed by their doctors after they experience CPAP in the laboratory during sleep.

14. **Unforeseeable Risks:** There are no potential risks that a person might suffer from resulting participation in this study.
15. **Injury/Illness:** If you have questions about this study or you experience any unexpected adverse event (side effect) while you are participating in this study, G. Vernon Pegram, Ph.D. or Mr. Jack Johnson should be contacted at (205) 599-1020 or (205) 934-7110. He will be available to answer your questions before, during, and after the study.
16. **New Findings:** If any significant new findings occur during the course of the study including problems that others who have participated have experienced in the study, you will be informed of this by one of the investigators listed above.
17. **How to Learn More About Your Rights as a Research Subject:**
If you have any questions about your rights as a research subject, contact the Baptist Health System Human Research Review Board, 800 Montclair Road, Birmingham, AL 35213, telephone 205/592-5700.
18. **Signatures:** The study has been discussed with me and all my questions have been answered. I may direct additional questions regarding study specifics to the investigators. I agree to participate in the study described above and acknowledge the investigator's obligation to provide me with a signed copy of the consent form.

Subject Signature

Date

The study subject has indicated to me that he/she is unable to read. I certify that I have read this consent form to the subject and explained that by completing the signature line above, the subject has agreed to participate.

Signature of Reader

Date

Appendix C

Consent Form for Participation in CPAP Adaptation, Form B

CONTROL GROUP

1. **Study Title:** Examination of a Method for Increasing Adjustment to and Compliance with Nasal Continuous Positive Airway Pressure (CPAP) in Persons with Obstructive Sleep Apnea Syndrome.
2. **Performance Sites:** Sleep Disorders Center of Alabama, Inc. and HealthSouth Medical Center Sleep Disorders Center
3. **Investigators:** Principal Investigator: Jack A. Johnson, Jr., MA. Mr. Johnson can be reached at (205) 982-9437 from 5:30 p.m. to 7:30 p.m.

Sub-Investigator: G. Vernon Pegram, Ph.D. Dr. Pegram can be reached at (205) 599-1020 or (205) 934-7110 from 8 a.m. to 5 p.m.
4. **Purpose of the Study:** To examine a method that may help patients to adjust better to nasal Continuous Positive Airway Pressure (called CPAP) treatment for sleep apnea.
5. **Subject Inclusion:** Any person age 19 or older who has been referred by his or her physician for a sleep study with CPAP (or with CPAP if indicated) is eligible for participation in this study. Only eligible persons have been invited to participate. Persons who have had a severe head or spinal injury that resulted in leakage of cerebrospinal fluid (the fluid that surrounds and cushions the brain) are not eligible to participate.
6. **Number of Subjects:** 92 Total People will participate in the experiment.
7. **Study Procedures:** All research subjects will be spending the night in the sleep laboratory for a diagnostic evaluation, which might include a CPAP trial aimed at finding a proper level of treatment (air pressure) for obstructive sleep apnea, a routine procedure for persons who have this sleep disorder. Some persons may not receive a CPAP trial during the course of the sleep study if they do not have obstructive sleep apnea or if the degree of their illness does not clinically require CPAP treatment. The diagnostic sleep study will occur immediately following each subject's participation in the research study. CPAP delivers a constant stream of pressurized air through your nose, which prevents you from stopping breathing during sleep. There will be no change in the way that CPAP is normally used in the the Sleep

Disorders Center of Alabama, Inc. or the HealthSouth Medical Center Sleep Disorders Center (depending on which sleep laboratory you are having your examination). You and others people in the group that you are participating in will receive the routine clinical care that is the current community standard of care in a sleep disorders center that has been accredited by the American Sleep Disorders Association. You will not be deprived of any preparations that are used to get people ready for their sleep studies. Data from your sleep study and home CPAP use (if you are prescribed CPAP) will be compared against persons receiving a variation of normal laboratory procedures, which is believed might make their adjustment to CPAP easier. All persons will receive written information. Your participation will not reduce or modify information that is normally gathered during the course of a sleep study. Finally, the proposed study is not an experimental procedure, but rather is a variation of the CPAP procedure, which has been designed, with the aim of making adjustment to CPAP easier.

8. **Benefits:** This study may benefit you in your adjustment to CPAP and will further scientific knowledge in the field of sleep medicine concerning the treatment of obstructive sleep apnea syndrome.
9. **Risks/Discomforts:** One risk is that some persons report experiencing anxiety, which may take the form claustrophobia, why wearing the CPAP mask. In general, there is only one major reason that CPAP should not be used for the treatment of sleep apnea. CPAP should not be used with persons who have suffered a very severe head or spinal injury that caused leakage of cerebrospinal fluid (the fluid which surrounds and cushions the brain). As mentioned above, persons who have had an injury of this nature are not eligible to participate.
10. **Right to Refuse:** You have the right to end your participation in this research study at any point that you choose and for any reason that you might have without penalty to yourself. Choosing to drop out of the study will not affect the treatment that you will receive during your sleep study tonight or at any other time regarding your sleep apnea.
11. **Privacy:** The results of this study may be published in a research paper, but your identity or the fact that you have participated in this study will never be made available to public or any other agency.
- Your participation in this research study will be kept confidential. However, during the study, representatives from the US Food and Drug Administration (FDA) will be allowed to review your

medical records that relate to the study. The Baptist Health System Human Research Review Board may also review these records. The records will identify you only by your initials and an assigned research study number, not by your name.

When you sign the consent document, you are giving your doctor permission to release your research study medical records for review. The representatives described in the paragraph above will use these records only to carry out their obligations associated with this study. Every effort will be made to ensure that your study records are not used for any other purpose, or given to anyone else, except when required by law.

12. **Financial Information:** You will not be paid to participate in this study, and there will be no cost to you for participating.

The Baptist Health System Inc, Sleep Disorder Center of Alabama, and Mr. Jack Johnson have made no provision for monetary compensation in the event of physical illness and/or injury resulting from this research study. In the event of such injury, medical treatment is available but is not free of charge. There is no monetary (or other) form of compensation for my participation in this study.

If you have any questions about compensation or medical treatment for research-related injuries, contact G. Vernon Pegram, Ph.D. or Mr. Jack Johnson at the Sleep Disorder Center of Alabama at 599-1020.

13. **Alternatives:** Currently, CPAP is the best treatment for sleep apnea for most persons. Your doctor is available to discuss other treatment options with you. The current study is not a treatment, but rather an examination of a way of helping people adjust to a treatment that they might be prescribed by their doctors after they experience CPAP in the laboratory during sleep.

14. **Unforeseeable Risks:** There are no potential risks that a person might suffer from resulting participation in this study.

15. **Injury/Illness:** If you have questions about this study or you experience any unexpected adverse event (side effect) while you are participating in this study, G. Vernon Pegram, Ph.D. or Mr. Jack Johnson should be contacted at (205) 599-1020 or (205) 934-7110. He will be available to answer your questions before, during, and after the study.

16. New Findings: If any significant new findings occur during the course of the study including problems that others who have participated have experienced in the study, you will be informed of this by one of the investigators listed above.

17. How to Learn More About Your Rights as a Research Subject:
If you have any questions about your rights as a research subject, contact the Baptist Health System Human Research Review Board, 800 Montclair Road, Birmingham, AL 35213, telephone 205/592-5700.

18. Signatures: The study has been discussed with me and all my questions have been answered. I may direct additional questions regarding study specifics to the investigators. I agree to participate in the study described above and acknowledge the investigator's obligation to provide me with a signed copy of the consent form.

Subject Signature

Date

The study subject has indicated to me that he/she is unable to read. I certify that I have read this consent form to the subject and explained that by completing the signature line above, the subject has agreed to participate.

Signature of Reader

Date

Appendix D

Alton Ochsner Medical Foundation Informed Consent

Examination of a Method for Increasing Adjustment to and Compliance with Nasal Continuous Positive Airway Pressure (CPAP) in Persons with Obstructive Sleep Apnea Syndrome

This study is not sponsored, and is a doctoral dissertation research project.

Principal Investigator: William F. Waters, Ph.D.

Sub-Investigators: Jack A. Johnson, Jr., MA and Mark Hurry, Ph.D.

You have been invited to participate in the clinical research study: Examination of a Method for Increasing Adjustment to and Compliance with Nasal Continuous Positive Airway Pressure (CPAP) in Persons with Obstructive Sleep Apnea Syndrome.

The doctors at Ochsner study the nature of disease and attempt to develop improved methods of diagnosis and treatment. This is called clinical research. To decide whether or not you should agree to be part of this research study, you should understand enough about its risks and benefits to make an informed judgment. This process is called informed consent.

Purpose

The purpose of this study is to examine a method that may help people to adjust better to nasal Continuous Positive Airway Pressure (called CPAP) treatment for sleep apnea. Sleep apnea is a sleep disorder which means that a person ceases to breathe repeatedly for a short periods of time while asleep. You have been asked to participate in this study because, based on a review of your symptoms, your physician has determined that you may be at significant risk of having sleep apnea. As part of your sleep study, you might receive CPAP treatment if professionals in the sleep laboratory determine that you have sleep apnea.

Procedure

You will be spending the night in the sleep laboratory for a diagnostic evaluation, which could include a CPAP trial aimed at finding a proper level of treatment (air pressure) for obstructive sleep apnea, a routine procedure for persons who have this sleep disorder. CPAP delivers a constant stream of pressured air through your nose, which prevents you from stopping breathing during sleep. You may not receive a CPAP trial during the course of the night if you do not have obstructive sleep apnea or if the degree of your illness does not warrant CPAP treatment during the same night as your initial diagnostic sleep study.

If you agree to participate in this research study, the diagnostic sleep study will occur immediately after we have completed the procedures required for this research study.

Subjects who agree to participate in this study will be randomly assigned to one of two groups. This means that you will have a 50/50 chance of being placed in either of these two groups. This method is similar to the flip of a coin.

If you are assigned to the first group, you will be asked to wear the CPAP mask with air pressure before bedtime while you are not in bed and not attempting to fall asleep. There will be no change in the way that CPAP is normally used in the Sleep Disorders Center, Ochsner Clinic, Baton Rouge. The total time that you will be asked to wear the CPAP mask with pressure is approximately 30 minutes, during which time you will experience a range of gradually building CPAP pressures that are commonly experienced during the course of a sleep study with CPAP. You will experience four pressure settings for exactly seven and a half minutes each. The pressure will be increased every seven and a half minutes and will increase a total of three times before the trial comes to a conclusion. During the time that you are wearing the CPAP mask, you may engage in any relaxing activity that you choose, including watching television or reading. Data from the sleep study and home CPAP use for the first several weeks you are using CPAP (only if you are prescribed CPAP treatment) will be compared against persons receiving the routine laboratory procedures.

If you are assigned to the second group, you will be asked to provide some additional information in a Sleep Disorders Inventory about your sleep problem in addition to receiving the routine clinical care that is currently the community standard of care in centers specializing in the diagnosis and treatment of sleep disorders. Data from your sleep study and home CPAP use for the first several weeks you are using CPAP (only if you are prescribed CPAP treatment) will be compared against those persons who are in the first group and are receiving a variation of the normal laboratory procedures.

You will receive written information about CPAP treatment for sleep apnea for educational purposes. Your participation will not reduce or modify information that is normally gathered during the course of a sleep study. Finally, the proposed study is not an experimental procedure, but rather is an examination of a variation of the CPAP procedure that has been designed with the aim of evaluating its potential in making adjustment to CPAP easier.

Benefits

This study may benefit you in your adjustment to CPAP and will further scientific knowledge in the field of sleep medicine concerning the treatment of obstructive sleep apnea syndrome with CPAP. No direct benefit to you may result from your participation in this study.

Risks

One risk is that some persons experience anxiety, which may take the form of claustrophobia, while wearing the CPAP mask. If this occurs, alert the investigator who will be in the room with you during the procedure. In general, there is only one major reason that CPAP should not be used for the treatment of sleep apnea. CPAP should

not be used in persons who have suffered a very severe head or spinal injury that caused leakage of cerebrospinal fluid (the fluid which surrounds and cushions the brain). Persons who have experienced an injury of this nature are not eligible to participate.

You understand that Louisiana state law requires that participants in all clinical studies such as this one be informed that any study (or procedure) may also result in death, brain damage, quadriplegia (paralysis in all arms and legs), paraplegia (paralysis of both legs), loss of organ, loss of arm or leg, loss of function of organ, loss of function of an arm or leg, and disfiguring scars.

Alternative Methods/Treatments

Currently, CPAP is clearly the best treatment for sleep apnea for most people. You do not have to participate in this study in order to receive CPAP. Your doctor will also discuss other treatment options with you, which range from weight loss to palatal surgery.

The current study is not a treatment for sleep apnea, but rather an examination of a method of helping people adjust to CPAP after they experience CPAP in the laboratory during sleep to prevent sleep apnea.

Payment for Participations and/or Reimbursement of Expenses

You will not be paid to participate in this study, and there will be no cost to you for participating.

Additional Costs

There are no known additional or anticipated costs for participating in the study.

Compensation for Injury

You understand that in the event of related injury from the research procedures, medical treatment, including hospitalization, if necessary for injuries or illness, is available. This medical treatment and/or hospitalization not provided free of charge. No provision for monetary compensation has been made in the event of an injury resulting from the research.

Confidentiality

The confidentiality of your records will be maintained to the extent consistent with the law. Governmental agencies, such as the Food and Drug Administration, may review and copy your records to ensure compliance with regulations and protocols. Additionally, the sponsor and Ochsner authorized officials may review your records to ensure compliance. You will not be identified in any reports or publications resulting from the study.

Contact Information

You understand that should you have any questions about the study or suffer any injury that you feel may be related to the study, you should contact the Responsible Investigator William F. Waters, Ph.D. or his associate, Jack A. Johnson, Jr., MA by calling (225) 761-5852. You also understand that you may take any problem or any question concerning your rights to the Office of Research Administration, Fifth Floor, Brent House, Telephone (504) 842-3562.

Safeguards

- . Whereas no assurance can be made concerning the results that may be obtained (since results from a clinical research study cannot be predicted), your physician, acting as Responsible Investigator, will take every precaution consistent with the best medical practice;**
- . By signing this consent form you have not waived any of your legal rights or released this institution from liability for negligence.**
- . Participation in this study is voluntary. You may revoke your consent and withdraw from this study at any time without any penalty or loss of benefits to which you are otherwise entitled;**
- . You have disclosed to your doctors all of your past and present diseases and allergies of which you are aware and all drugs and medications which you are presently using.**

Additional Information

If any significant new findings occur during the course of the study, including problems that others who have participated have experienced in the study, you will be informed of this by one of the investigators above. Although not anticipated, the investigators reserve the right to eliminate any subject from participating further regardless of a subject's consent. Circumstances under which this might occur would include terminating the CPAP trial early if continuation of the trial would prevent you from starting your sleep study on time during the night of your participation. There are no consequences of any kind for a subject who wishes to voluntarily withdraw from the study. The procedure for withdrawing from the study is to 1) inform the investigator during the course of your participation during the experiment or 2) to contact one of the investigators at the telephone number listed above if you wish to withdraw at a later time.

Statement of Voluntary Agreement to Participate

The procedure(s) involved, expected duration of participation, alternative methods/treatments, and possible benefits, discomforts, risks, and adverse effects have been explained to me in language I understand as set forth above by Dr. William F. Waters or his representative, Jack A. Johnson, Jr., MA. I have been given the time to ask questions, which have been answered to my satisfaction. I have been given a copy of this consent for my information and records. I voluntarily consent to participate in this investigation.

Patient

Date

Patient ID Number

Witness

Date

Physician (Responsible Investigator)

Date

Appendix E

Sleep Disorders Inventory

William F. Waters, Ph.D., ABPP, BCSS
Director, Ochsner Clinic of Baton Rouge, Sleep Disorders Center
and
Professor, Department of Psychology, Louisiana State University

Please provide the following information:

Name: _____ Clinic Number _____
Date: _____ Age: _____ Birth Date: _____ Weight: _____
Height: _____ BMI _____

Instructions: The following questions will help us understand any sleep problems you may have. Please answer all of the questions to the best of your ability. For some questions, you should *circle* YES if the item is true for you or NO if the item does not apply to you. For other questions, a space is provided for you to write a number, such as how many minutes it takes you to fall asleep. For all questions, give an answer that is the closest to the truth as you know it. For some questions, you may have to ask someone who has seen you sleep; if no one has seen you sleep, write DK (don't know).

1. What is your primary sleep problem?

How long have you had that problem? ____ No. Years; ____ No. Months; ____ No. Weeks

2. At what time do you usually turn out the lights to go to sleep? _____
At what time do you usually wake up for the next day? _____

3. How many minutes does it usually take you to fall asleep? _____

4. How many nights a week do you get 9 or more hours sleep? _____
How many nights a week do you get 8 hours sleep? _____
How many nights a week do you get 7 hours sleep? _____
How many nights a week do you get 6 hours sleep? _____
How many nights a week do you get 5 or less hours sleep? _____

5. Do you take 30 minutes or more to fall asleep, more than once a week? YES NO
If YES: How many nights does this happen each week? _____
On nights when you have this problem how many minutes does it take you to fall asleep? _____
On nights when you have this problem, how many hours do you sleep? _____

6. Do you wake up during the night and take 20 minutes or more to regain sleep, more than once a week? YES NO

- If YES:** How many nights does this happen each week? _____
 On average, how many times does this happen each night? _____
 How many minutes does it take you to fall back asleep each time? . _____
 On nights when you have this problem, how many hours do you sleep? _____
7. Do you often wake up in the morning before your scheduled wake time, and cannot go back to sleep? YES NO
If YES: How many nights each week do you have this problem?
 On nights when you have this problem, how many hours do you sleep? _____
8. Do you often fall asleep at inappropriate times or places during the day because you are not getting enough sleep? YES NO
9. Do you often have trouble functioning during the day because you are not getting enough sleep? YES NO
10. On nights when you do get a full night's sleep, do you still:
- Have trouble waking up, or wake up feeling unrefreshed? YES NO
 - Fall asleep involuntarily during the day, but only when somewhat unstimulated? YES NO
If YES, check each example that applies to you: While watching TV _____ ; while reading a book _____ ; while a passenger in a car _____ ; while in a traffic jam _____ .
 - Fall asleep involuntarily during the day, even when doing something important or stimulating? YES NO
If YES, check each example that applies to you: While driving _____ ; while doing your work _____ ; while talking to others _____ .
 - Have trouble functioning during the day? YES NO
11. Do you ever sleep 12 or more hours and still wake up unrefreshed? YES NO
If YES, do you still need to nap during the day? YES NO
If YES, how many times a month does this occur? _____
12. How many nights per month do you:
 _____ Snore loudly and persistently
 _____ Thrash about while asleep (but are not dreaming)
 _____ Gasp or snort while asleep
 _____ Stop breathing while asleep or wake up in the night and feel unable to breathe
 _____ How many mornings a month do you awaken with a headache?
13. Does it often take you longer to fall asleep because your legs feel restless or odd in bed? YES NO
If YES: Does moving your legs in bed, or getting up and moving around help you fall asleep? YES NO
 How many nights per month do your legs feel this way? _____

14. Do you often wake from a sound sleep repeatedly because your legs jerk? . . . YES NO
 If YES: Are your leg movements frequent and regular? YES NO
 How many nights per month does this happen? _____
15. Have sleep attacks in which you suddenly and uncontrollably fall asleep? . . . YES NO
 If YES: How many minutes do you sleep (nap) when you have such an attack? _____
 Do you awaken from your nap feeling refreshed? YES NO
 How many times per month does this happen? _____
16. When you are startled, emotional, excited or happy do you often experience extreme weakness (for example, in your legs) or drop things? YES NO
17. When you are startled, emotional, or excited do you often collapse or fall? . . YES NO
 If YES: Are you still aware of your surroundings? YES NO
18. As you fall asleep or wake up, do you often see things that are not there? . . . YES NO
 If YES: Are the things you see very clear and realistic? YES NO
 How many times each month does this happen? _____
19. As you fall asleep or wake up, are you often unable to move (paralyzed)? . . . YES NO
 If YES: How many times each month? _____
18. How many nights have nightmares awakened you in the last month? _____
 If ANY: How intense are they? (1=Mild, 2=Frightening, 3=Terrifying) _____
19. Do you often move violently during your sleep while dreaming, and sometimes even hurt yourself or your partner by accident or fall out of bed? YES NO
 If YES: How many times in the last month has this happened? _____
20. Do you often wake up from a deep sleep sweating, your heart beating fast or pounding, with a feeling of fear but with no memory of a dream? YES NO
 If YES: How many times in the last month has this happened? _____
 How intense is this experience? (1=Mild, 2=Frightening, 3=Terrifying) _____
 Does this ever happen during the day? YES NO
21. Do you often grind your teeth in your sleep? YES NO
22. How many times each night do you wake up specifically to use the bathroom? _____
23. How many nights each week do you wake up with indigestion or heartburn? _____
24. Do you often eat your last meal or a large snack within 2 hours of bedtime? . YES NO
25. Do you often exercise vigorously within an hour of bedtime? YES NO
26. How many nights each month do you use alcohol within 2 hours of bedtime. _____

27. How many nights each month do you use alcohol to help you fall asleep? . . . _____
28. How many caffeinated beverages do you drink in a day? _____
29. How many days a week do you drink caffeinated beverages after 7 p.m.? . . . _____
30. How often is your sleep problem caused or made worse by physical discomfort or pain? (check one): Never _____ Rarely _____ Sometimes _____ Often _____
Most or All of the Time _____
31. Do you ever work night shifts (any 8-12 hour shift starting after 6 pm)? YES NO
If YES: How many nights per month? _____
32. Do you often work at home after 8 pm? YES NO
If YES: How many nights per week? _____
33. Do you deliberately sleep less in order to do other things? YES NO
If YES: How many nights per week? _____
How many hours per night? _____
34. On weekends or your days off, do you often sleep more than 1 hour later than your usual wake up time? YES NO
35. Do you often go to bed earlier to make up for lost or unrefreshing sleep? . . . YES NO
36. Do you often wake up later to make up for lost or unrefreshing sleep? YES NO
37. Do you take naps? YES NO
If YES: How many times each week do you take naps? _____
How many minutes are your naps, on average? _____
Do you awaken from your naps refreshed? YES NO
38. Do you often lose sleep because your bed partner disturbs you at night? YES NO
39. Is your sleep often disturbed by environmental factors, such as traffic, neighbors or family members? YES NO
40. Do you often lose sleep because your bedroom is not dark enough at night? . YES NO
41. Do you often lose sleep because your bedroom temperature is not comfortable enough at night? YES NO
42. Do you usually sleep better when you sleep away from home? YES NO
43. When you try to sleep, does worrying or problem solving often keep you awake? . . .
. YES NO

44. Do you often worry, in bed, about getting enough sleep to function the next day? YES NO

45. Do you often get frustrated and angry, in bed, about not getting to sleep? . . . YES NO

46. Do you worry too much in general? YES NO

47. Have you been under noteworthy stress recently? YES NO

48. Check if you are currently diagnosed with: ____ depression ____ an anxiety disorder

49. Have you recently taken any prescription or over-the-counter medication for sleep problems? YES NO

If YES: How many nights a week do you usually take this medication? . . . _____

How many months have you been taking this medication? _____

50. Do you take any medications that contain caffeine or other stimulants, such as allergy medications, nasal decongestants, or pain killers? YES NO

If YES: How many minutes or hours before trying to sleep do you take them? _____

51. Please list all prescription and over-the-counter medications you are now taking, and what each is for:

MEDICATION	CONDITION

MEDICATION	CONDITION

52. Please list any medications you have recently stopped taking, and what each was for:

MEDICATION	CONDITION

MEDICATION	CONDITION

You may write any additional information that you think could be helpful in the space below, or on the back of this page:

Appendix F

Correlation Coefficients for PSG Variables: Split-Night

Variable	SOL	SE	%1	%SWS	REM	STA	No. Awake
SOL	—	-.56**	.41**	-.17	-.25	.49**	.15
SE		—	-.52**	-.02	.32*	-.45**	-.53**
%1			—	-.19	-.25	.74**	.62**
%SWS				—	-.02	-.11	-.19
REM					—	-.22	-.19
STA						—	.44**
No. Awake							—

Note. * significant at .05 level, 2-tailed, ** significant at .01 level, 2-tailed.

Appendix G

Correlation Coefficients for PSG Variables: Full-Night

Variable	SOL	SE	%I	%SWS	REM	STA	No. Awake
SOL	—	-.59**	.42**	-.23	-.53*	.39**	.21
SE		—	-.51**	.18	.44	-.33**	-.58**
%I			—	-.31*	-.56*	.69**	.67**
%SWS				—	.28	-.14	-.33*
REM					—	-.04	-.53*
STA						—	.42**
No. Awake							—

Note. * significant at .05 level, 2-tailed, ** significant at .01 level, 2-tailed.

Appendix H

Correlation Coefficients Relevant to an Examination of CPAP Use

Variable	Gender	BMI	A+HI	Accept	CPAP Use	Titration
Gender	---	-.01	-.36**	.06	-.33*	.54**
BMI		---	.21	.01	.16	-.14
A+HI			---	.11	.22	-.40**
Accept				---	---†	.05
CPAP Use					---	-.32*
Titration						---

Note. * two-tailed significance of $p \leq .05$, ** two-tailed significance of $p \leq .01$, † cannot be calculated because Use is dependent upon Acceptance of CPAP.

Vita

Jack A. Johnson, Jr. was born in Atlanta, Georgia and grew up in a suburb Knoxville, Tennessee. He lived in Knoxville until he was 24 years old and spent too much time playing guitar and Super Nintendo with friends from college. Jack moved to Baton Rouge in the fall of 1994 to begin his graduate studies in psychology at the Louisiana State University and Agricultural and Mechanical College. He also spent two long and boring years in Birmingham, Alabama where he and his wife, DeAnn M. Johnson, completed psychology internships at the University of Alabama at Birmingham psychology consortium. As you probably guessed because he is from the southern United States, Jack is a rabid college football fan and follows the Tennessee Volunteers and the Tigers almost every Saturday from September through the first week of January. Additionally, Jack and his wife enjoy the New Orleans Saints' games on Sundays. You might be surprised to know that Jack's true passion in life is not psychology but is history. Jack is particularly interested in the sacrifice and courage of those who fought in World War II. American history is another area of great interest for Jack, and he plans to spend much of his free time reading about noteworthy events in history. Also, Jack has aspirations to write about history, and has plans to explore writing a book about men from Louisiana who served in the United States Merchant Marines during World War II. Music is another area of interest for Jack, and he wrote the passage you are now reading while listening to the Red Hot Chili Peppers' Blood Sugar Sex Magic. Aerosmith, AC/DC, and Led Zeppelin are long time favorites of his and helped ease the pain of completing graduate school. Finally, Jack's life history would not be complete without mentioning his loyal and loving cat Claire, known to her friends as Swiggums. She particularly likes spending quality time with her family, and is generally one cool cat.


DOCTORAL EXAMINATION AND DISSERTATION REPORT

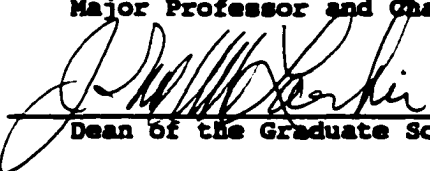
Candidate: Jack Allen Johnson, Jr.

Major Field: Psychology


Title of Dissertation: Examination of a Method for Increasing Adjustment to and Compliance with Nasal Continuous Positive Airway Pressure (CPAP) in Persons with Obstructive Sleep Apnea Syndrome


Approved:

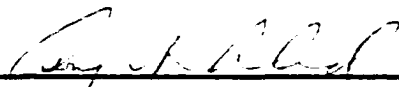

Major Professor and Chairman

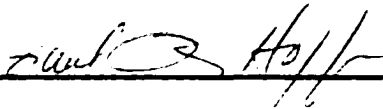

Dean of the Graduate School

EXAMINING COMMITTEE:









Date of Examination:

October 24, 2001